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RECENT DEVELOPMENTS IN THE DIAGNOSIS OF CONGENITAL HEART DISEASE*

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Fifty years ago Maude Abbott's first paper on heart disease[1] presaged a lifetime of study culminating in her familiar clinico-anatomic classification of congenital heart disease.[2] Ten years ago, one year before Abbott's death, the first successful ligation of a patent ductus arteriosus was carried out.[3] Four years ago an operation producing mitigation of the cyanosis caused by the tetralogy of Fallot and its analogues was reported,[4] and in the same year there were announced surgical cures of coarctation of the aorta[5, 6] and of vascular rings obstructing the trachea and esophagus.[7] Approximately a third of children born with congenital heart disease may now anticipate amelioration or cure of their condition through surgery. This statement is possible not only because of the vision and technical skill of surgeons such as Gross and Blalock, but because of the pioneer work of Abbott, the contributions of many to the progress of thoracic surgery, anesthesia, and preoperative and postoperative care, a more precise understanding of the altered physiology of congenital heart disease, the development of new and improved diagnostic methods, and finally accurate clinical appraisal, a field in which the name of Taussig is pre-eminent.[8]

A cardiovascular study program has been in progress at Charity Hospital in New Orleans for the past year. This communication is a report on some phases of the study, indicating in particular the application of the technics of arterial puncture and venous catheterization.

The study of arterial blood of human beings, first carried out almost forty years ago,[9] has during the past decade become an integral part of the clinical evaluation of pulmonary and cardiac disease. In the diagnosis of congenital heart disease it is the oxygen content of the arterial blood that is of chief importance. This may be precisely measured by actual gasometric analysis of blood samples, or somewhat less accurately by means of a photoelectric device clipped to the lobe of the ear.[10]

In the patient with congenital heart disease there are two mechanisms which may lower the oxygen saturation of the arterial blood. One is thickening of the alveolar-capillary membrane in the lungs, with consequent sufficient retardation of the rate of diffusion of oxygen from the alveoli into the capillaries to prevent blood passing through the lungs from becoming fully arterialized. Cyanosis due to such impaired pulmonary diffusion of oxygen is of infre-

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quent occurrence in congenital heart disease but is a potential threat to every patient with uncomplicated septal defect or patency of the ductus arteriosus. This type of unsaturation is completely overcome when the patient breathes pure oxygen.

Arterial oxygen unsaturation is more commonly caused by abnormal shunting of venous blood into the systemic arterial circulation without ever passing through the lungs. The prototype of such shunts is the tetralogy of Fallot. In this anomaly blood entering the right atrium from the great veins passes into the right ventricle, whence only a part passes through the stenotic pulmonary valve. The remaining blood is shunted into the overriding aorta, there to mix with normally oxygenated blood which has come from the lungs, and the net result is unsaturation of the blood in the systemic arteries. By the technic of angiocardiography it is possible to visualize the intracardiac shunt in the tetralogy of Fallot.

It is clear that arterial oxygen unsaturation which is due to a shunt cannot be overcome by the inhalation of oxygen because of the ever-present admixture of venous blood with arterial. Administration of 100 per cent oxygen during the withdrawal of arterial blood thus permits the distinction to be made between the two factors that produce arterial anoxemia.

Arterial unsaturation, be it due either to impaired pulmonary diffusion of oxygen or to a right-to-left shunt, is aggravated by muscular exercise. One may thus obtain an objective measure of exercise tolerance by leaving the arterial needle in place while the patient exercises, and withdrawing samples before and immediately after exercise.

From the oxygen content of blood in the right atrium, the pulmonary veins and a systemic artery, the actual fraction of blood shunted across the septal defect can be calculated.[11] Arterial oxygen content can be estimated directly; pulmonary venous oxygen can be calculated from readily available data with a fair degree of precision; while for right atrial oxygen content, one may use an assumed average value or actually remove with a catheter a sample for analysis.

Catheterization of the human heart is a technic that has been known since a German surgeon tried it out on himself twenty years ago.[12] His first attempt, in which a colleague inserted a ureteral catheter through a large needle into a left antecubital vein, was terminated because of the colleague's apprehension when the catheter tip lay in the axillary vein. Forssmann undertook the procedure by himself a week later, and after cutting down on a left antecubital vein passed the 65 cm. long catheter as far as it would go, verified its position in the right atrium by observing his fluoroscopic image in a mirror held by a nurse, and took an x-ray which is reproduced in his report. "The length of the catheter did not permit its further advance," the author observed. If he had been able to employ one of the 100 cm. long catheters now used for the purpose he might have observed it to pass down into the inferior cava and perhaps into a hepatic or renal vein, into the coronary sinus, or into the right ventricle and thence out into the pulmonary artery, where the vigorously oscillating catheter tip would have been seen to lie free of the heart shadow in the right or left lung field.

The technic of catheterization of the human heart was revived by Cournand and Ranges[13] some twelve years after Forssmann's experiments, and the procedure has now become commonplace. Though more time-consuming than arterial puncture, it requires less skill for its successful consummation. Procaine is infiltrated into the skin over an antecubital vein; we occasionally use general anesthesia in infants and small children. The vein is surgically isolated and the catheter is passed into a hole in its wall and thence up the arm. Further progress of the catheter is carried out under direct fluoroscopic observation. By trial and error and rotation of its inclined tip it is guided successively into the right atrium, right ventricle, pulmonary artery, and one or both of the pulmonary artery

branches. Then the infusion of 5 per cent glucose is intermittently interrupted to permit the blood pressure to be taken with an electrical manometer and blood samples to be removed for analysis of their oxygen content. These operations are repeated in each chamber as the catheter is withdrawn. At the termination of the procedure the catheter is removed from the arm, the skin edges are approximated with two or three sutures, and a pressure dressing is applied. Healing of the vein and return of its function are anticipated in the great majority of instances.

Three observations of importance are thus possible through the employment of this procedure. In the first place, any abnormality of pressure, be it high or low, may be detected. The most striking abnormalities in pressure are seen in cases of pulmonic stenosis, in which the pressure, abnormally low in the pulmonary artery, suddenly becomes abnormally high as the catheter tip is drawn back through the pulmonary valve into the right ventricle.

Secondly, any shunt of oxygenated blood into the venous circulation through an abnormal communication will be indicated by a rise in the oxygen content of the blood at a point opposite the shunt and beyond. Thus, if a defect in the atrial septum permits arterialized blood from the left atrium to flow into the right, the blood in the latter chamber will be found to have a higher oxygen content than samples removed from the venae cavae, and this increased oxygen content will be found to be present also in right ventricular and pulmonary arterial blood. If the left-to-right shunt occurs through a ventricular septal defect the rise in oxygen content takes place in the right ventricle and the pulmonary arterial content is also high. In the case of patent ductus arteriosus the oxygen contents of caval, right atrial, and right ventricular blood samples are the same, but an abrupt rise occurs in the pulmonary artery, due to shunting of blood from the aorta at this point.

Whenever an uncomplicated septal defect or patent ductus arteriosus is present the shunt is from left to right and the

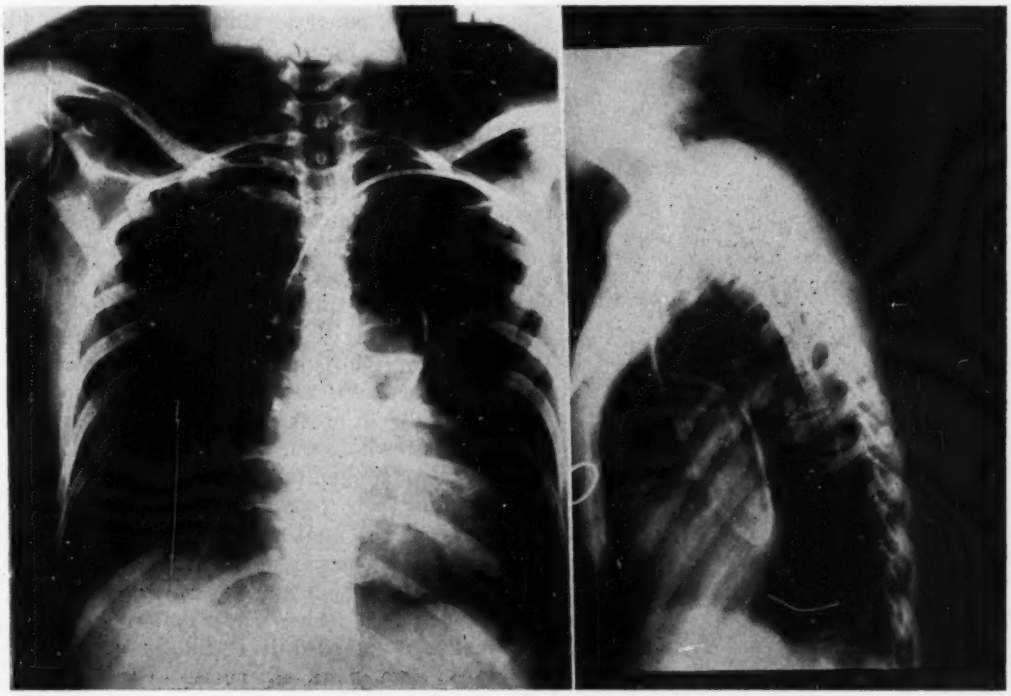

Figure 1. Anteroposterior and lateral x-rays of patient with atrial septal defect.

pulmonary blood flow is thereby increased. An enormous burden may thus be placed on certain chambers of the heart and on the pulmonary blood vessels, a burden which may result in the thickening of the alveolar-capillary membrane to which reference was made earlier. In a manner analogous to the calculation of right-to-left shunt in the tetralogy of Fallot the magnitude of a left-to-right shunt may be calculated when one knows the oxygen content of the arterial blood and of samples removed from the right side of the heart at points proximal and distal to the location of the shunt.[14]

Finally, when a septal defect is present one sometimes has the good fortune to observe the catheter actually traverse the defect and enter into the systemic side of the circulation. In Figure 1 the catheter is seen to have entered the left atrium directly from the right and to have further progressed into a left pulmonary vein. Figure 2 is the roentgenogram of a young man with the tetralogy of Fallot. The catheter has passed from the right ventricle into the overriding, dextraposed aorta, and its further course has been around the circle of the arch and down the thoracic aorta; its tip is seen to lie below the dome of the diaphragm.

Figure 2. Lateral x-ray of patient with the tetralogy of Fallot.

Complete study of the patient with congenital heart disease includes history, physical examination, roentgenographic and roentgenoscopic examination, and electrocardiographic examination. While arterial puncture and cardiac catheterization are valuable adjuncts in the precise evaluation of congenital heart disease, they occupy a place of no greater importance than the steps in the older, familiar diagnostic routine. The results of the newer tests can be interpreted only in the light of the basic data supplied by the other procedures.

The cooperative nature of this study program deserves emphasis. The authors of this group of papers represent the Departments of Surgery, Medicine, and Pediatrics at Louisiana State University School of Medicine, the Heart Station, and resident physicians' staff at Charity Hospital, and the United States Public Health Service. We have had the active cooperation of the Departments of Radiology and Anesthesiology at Charity Hospital, and acknowledge particular indebtedness to the two devoted technicians whose working day ends only when the analyses are completed. Once a week the results of recent studies are reviewed in informal conference attended by any and all interested persons, including members of the Pediatric and Medical staffs of the Tulane University School of Medicine. We also acknowledge the important role of the physicians throughout the state who, after making the initial diagnosis of congenital heart disease, are referring their patients to us for further study. The spirit of cooperative endeavor is carried over into the treatment of patients for whom surgery is indicated. In an age in which sovereign rights are jealously guarded it is indeed refreshing to be part of an enterprise in which such rights are subordinated to progress of the

cardiovascular study program in general, and to the welfare of the patient in particular.

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APPLICATION OF NEWER DIAGNOSTIC METHODS IN CONGENITAL HEART DISEASE*

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Modern cardiovascular surgery has improved so rapidly in the past fifteen years that we may no longer be satisfied merely to make the diagnosis of congenital heart disease. More accurate anatomical and physiological diagnoses must be obtained in order to determine operability. Surgery is facilitated when exact positions of septal defects and of great vessels are known, and the pressure relationships in these vessels and chambers has been determined.

In the past year, the cardiovascular unit at Charity Hospital has studied 98 cases, performing 74 cardiac catheterizations.‡ The purpose of this communication is to present 4 cases, each representative of one of the more common types of anomalies which we have encountered. The diagnoses include patent ductus arteriosus, interatrial septal defect, interventricular septal defect, and the tetralogy of Fallot.

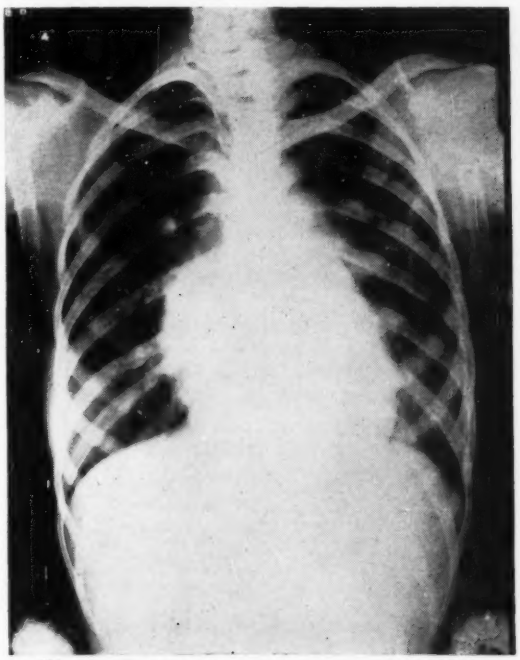

Fig. 1. Erect postero-anterior x-ray of chest of patient M. D. Cardiac enlargement and prominent vascular markings.

I. PATENT DUCTUS ARTERIOSUS

M. D., a colored female, age 19 years, had known of "a big heart" since the age of five; however, she had been completely asymptomatic until Jan-

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‡These studies have been made possible by the co-operation and generous support given to the Heart Station by the Administrators of Charity Hospital.

uary 1949, when she developed chills and fever.

Physical examination revealed a blood pressure of 124/58 in both arms and temperature 103° F. There was bulging of the precordial region of the chest wall. The heart was enlarged, by percussion, to the right and left. At the second left intercostal space near the sternal border there was a continuous machinery-like murmur with systolic accentuation.

The red blood cell count was 3 million per cu. mm.

X-ray and fluoroscopic examinations showed marked right and moderate left ventricular enlargement, with a prominent pulsating pulmonary conus and artery. (Fig. 1.)

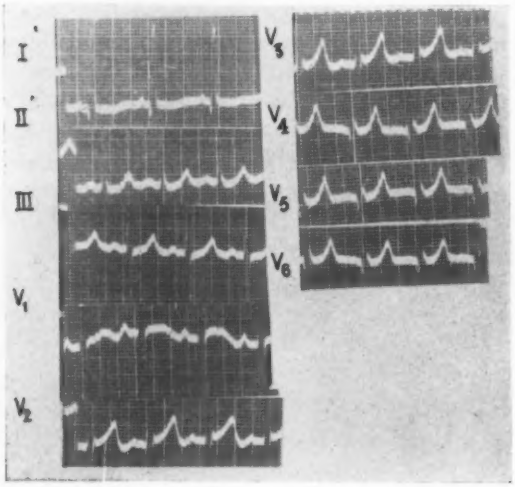

Fig. 2. Electrocardiogram of patient M. D. Left axis deviation with prominent R wave in leads I and VI. The peaks of the R waves were cut off when tracing was mounted.

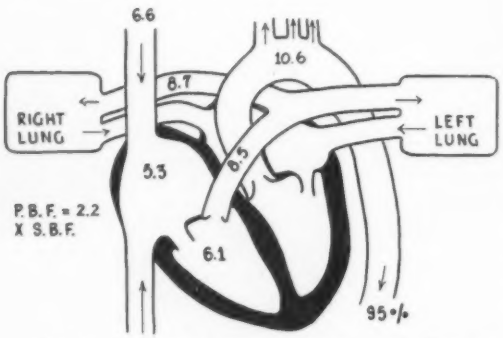

Fig. 3. Schematic representation of findings at cardiac catheterization and peripheral arterial oxygen saturation of patient M. D. Oxygen analyses were performed by a modified Roughton-Scholander micro-method. The 95% figure represents the percentage of oxyhemoglobin (normal 91-96%). The other figures are oxygen content of whole blood expressed in vol. per 100 cc.

Electrocardiogram was compatible with right and left ventricular hypertrophy. (Fig. 2.)

Cardiac catheterization confirmed the presence of a patent ductus arteriosus. There was an increase of 2.4 volumes per 100 cc. in the oxygen content of blood secured from the pulmonary artery as compared with that from the right ventricle. Arterial oxygen saturation was normal. (Fig. 3.) A beta-hemolytic streptococcus was isolated from the pulmonary arterial blood secured at the time of catheterization, confirming the clinical impression of bacterial endarteritis.

Pressure in the right atrium, ventricle, and pulmonary artery was elevated, especially the diastolic pulmonary artery pressure. (Fig. 4.) Calcula-

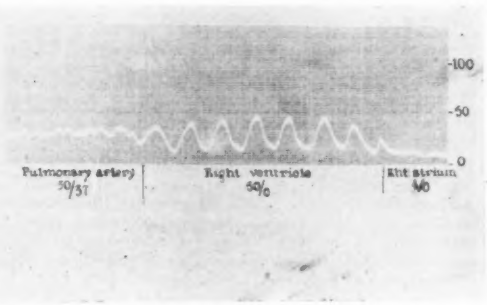

Fig. 4. Pressure tracings obtained at the time of cardiac catheterization of patient M. D., employing a Statham strain gauge manometer connected to an electrocardiographic apparatus.

tions carried out as indicated in the previous paper showed the pulmonary blood flow to be 2.2 times the systemic blood flow.

The patient received 10,000,000 units of penicillin daily and has been afebrile since the eighteenth day of therapy.

II. INTERATRIAL SEPTAL DEFECT

A. I., 58 year old colored female, was asymptomatic until the age of 48 years when she first experienced congestive heart failure. At that time she was found to have a blood pressure of 155/100, and "very large heart" with a regular sinus rhythm. There was a loud, harsh, systolic murmur, heard best at the fifth left intercostal space near the sternum, and transmitted toward the axilla. For the last ten years she had fluctuated between regular sinus rhythm and auricular fibrillation. During this time she was in congestive failure at intervals varying from six months to two years.

On present admission, x-ray and fluoroscopic examinations showed marked right and left ventricular enlargement, prominent pulsating pulmonary vessels and conus, and no evidence of left atrial enlargement. (Fig. 5.)

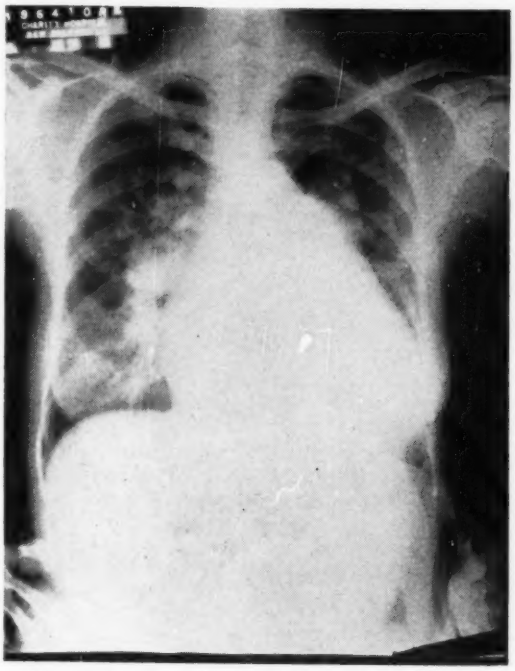

Fig. 5. Erect postero-anterior x-ray of chest of patient A. I. Marked cardiac enlargement to right and left with prominent pulmonary arteries and increased prominence of vascular shadows.

The electrocardiogram was compatible with right ventricular hypertrophy. (Fig. 6.)

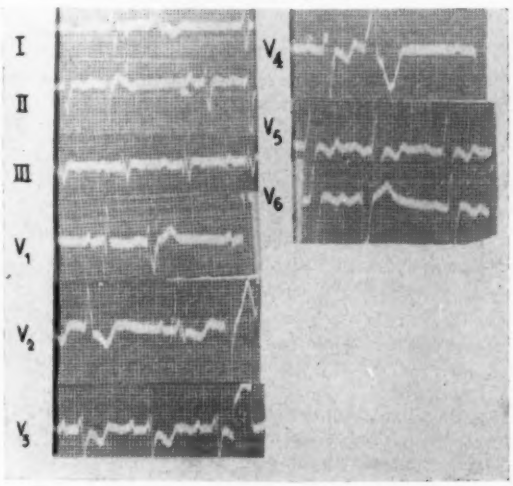

Fig. 6. Electrocardiogram of patient A. I. Compatible with right ventricular hypertrophy.

Analysis of blood secured at the time of cardiac catheterization revealed a rise in oxygen content of 5.9 volumes per 100 cc. in the right atrial blood as compared with the superior vena cava, confirming the presence of an interatrial septal defect with a left-to-right shunt. The catheter was passed through the defect into the left atrium and thence into a left pulmonary vein. Blood secured from the pulmonary vein was 97 per cent saturated with oxygen while the arterial blood was only 76 per cent saturated with oxygen (Fig. 7). These

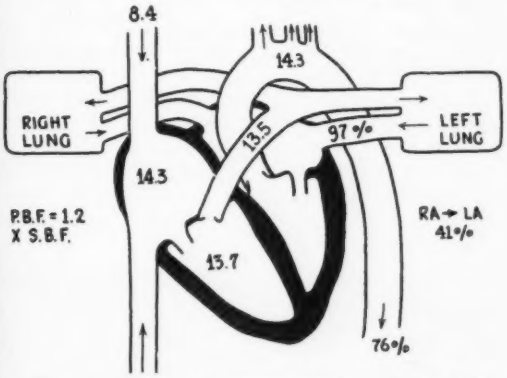

Fig. 7. Schematic representation of the findings at cardiac catheterization and peripheral arterial oxygen saturation of patient A. I.

observations indicate that there is a right-to-left shunt through this defect as well as a left-to-right shunt. The right-to-left shunt, opposite to the pressure gradient, may be explained by turbulence of rapidly moving blood with passage of some right atrial blood through a large defect into the left atrium. Forty-one per cent of the systemic venous return to the heart is calculated to be shunted into the left atrium in this manner. The left-to-right shunt, following the pressure gradient, is such that the pulmonary blood flow is calculated to be 1.2 times the systemic blood flow.

There was a marked elevation in pressure in all chambers. Systolic and diastolic pressures in the left atrium were higher than that found in the right atrium. (Fig. 8.)

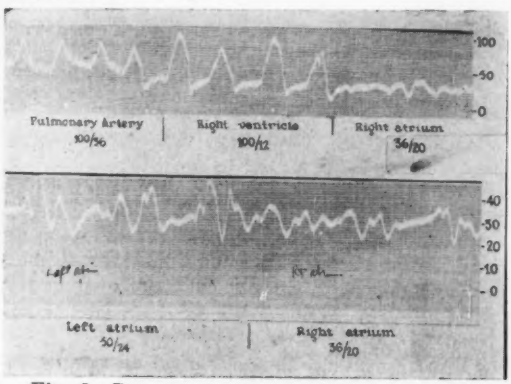

Fig. 8. Pressure tracings obtained at the time of cardiac catheterization of patient A. I. Tracings made with manometers of different sensitivity.

III. INTERVENTRICULAR SEPTAL DEFECT

S. S. C., a 7 year old white female, was known to have heart trouble at birth. Growth and development were normal, and there was no exercise intolerance, cyanosis, or clubbing. At four years of age, she was successfully treated with penicillin at Charity Hospital for subacute bacterial endocarditis.

Physical examination revealed blood pressure of 112/76, moderate enlargement of the heart to the left and right, and a loud, harsh holosystolic murmur heard best at the fourth left intercostal space near the sternum. Red blood cell count was 4.5 million per cu. mm.

X-ray and fluoroscopic examinations showed slight right and left ventricular enlargement with prominence of the pulmonary conus and pulmonary vessels. (Fig. 9.)

Fig. 9. Erect postero-anterior x-ray of chest of patient S. S. C., showing prominence of pulmonary artery and some right and left ventricular enlargement.

Electrocardiogram was within normal limits. (Fig. 10.)

Analysis of blood secured at the time of catheterization revealed the evidence of an interventricular septal defect. Blood secured from the right ventricle had 1.5 volumes per 100 cc. more oxygen than that of the right atrium. (Fig. 11.) The pressures were somewhat elevated in the pulmonary artery and right ventricle.

The arterial blood had a normal oxygen saturation.

The shunt causes the pulmonary blood flow to be about 1.4 times the systemic flow. In our experience this is considered to be of a small order, and probably signifies a favorable prognosis.

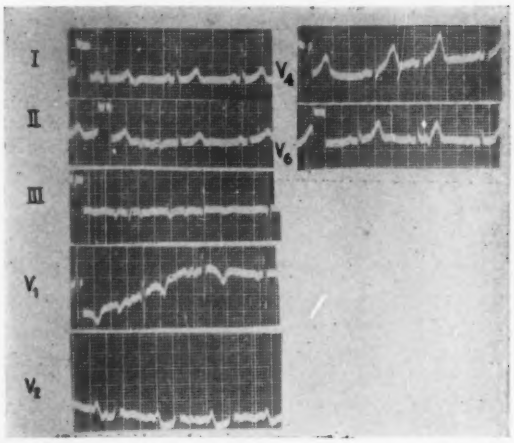

Fig. 10. Electrocardiogram of patient S. S. C. showing sinus arhythmia. Electrocardiogram within nomal limits.

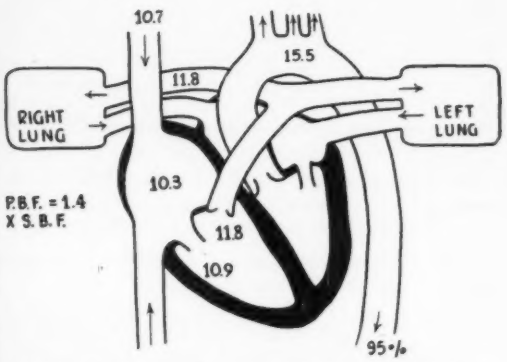

Fig. 11. Schematic representation of findings at cardiac catheteriztion and peripheral arterial oxygen saturation of patient S. S. C. Pressure in the pulmonary artery was 23 mm. Hg. (mean), in the right ventricle 21 mm. Hg. in the right atrium 4 mm. Hg.

IV. TETRALOGY OF FALLOT

J. C., a seven year old white female, developed cyanosis and clubbing of the fingers and toes at one year of age. She had progressive exercise intolerance.

Physical examination revealed poor development and nourishment, cyanosis, and clubbing of the fingers. Blood pressure was 90/60; there was a marked precordial bulge, and the heart was enlarged to the right and left on percussion. A harsh holosystolic murmur was heard at the third left intercostal space near the sternum, radiating throughout the precordium.

She had 7.73 million red blood cells per cu. mm.

X-ray and fluoroscopic examinations gave evi-

dence of right ventricular hypertrophy; the pulmonary conus area was concave, however, and the peripheral pulmonary markings were decreased, suggesting diminished pulmonary blood flow. (Fig. 12.)

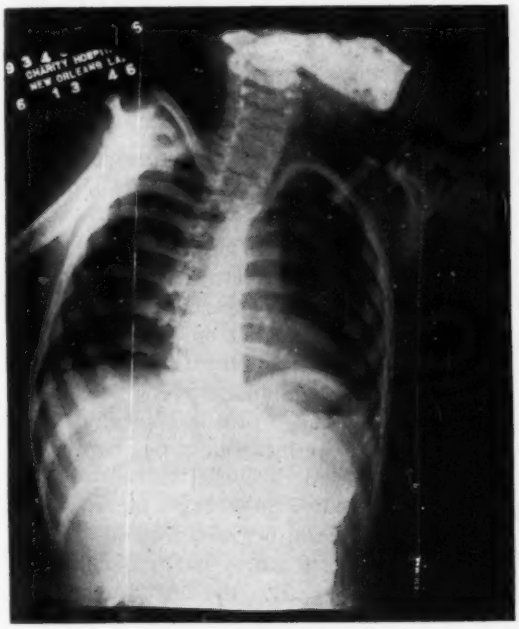

Fig. 12. Right anterior oblique view of patient J. C., showing concave pulmonary conus region with cardiac enlargement to the right and clear lung fields.

Electrocardiogram showed right ventricular hypertrophy. (Fig. 13.)

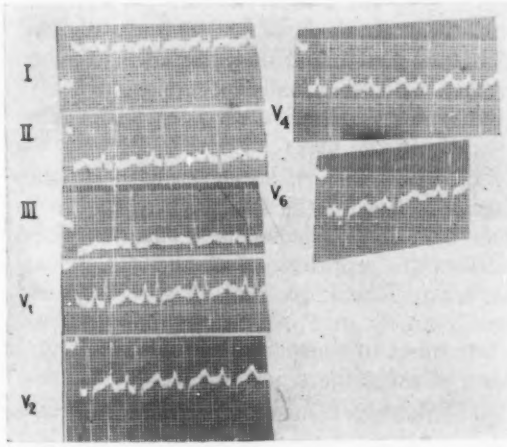

Fig. 13. Electrocardiogram of patient J. C., showing marked right axis deviation with a prominent R in V1 and S in V6.

The oxygen content of blood secured from various cardiac chambers by catheterization gives no conclusive evidence of left to right shunting. Since systemic arterial oxygen saturation of 75 per cent did not rise to 100 per cent during the inhalation of oxygen, a shunt from the right ventricle into the systemic circulation is indicated. Calculation showed this to be approximately 60 per cent of the venous return.

The extremely low pressure in the pulmonary artery was in sharp contrast to the high right ventricular pressure. This finding established the presence of pulmonary stenosis. The identity of right ventricular and systemic arterial systolic pressures suggest the presence of a direct communication between the right ventricle and aorta. The aorta therefore arises in part from the right ventricle. (Fig. 14.)

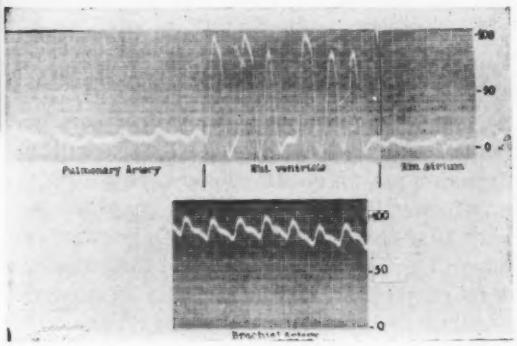

Fig. 14. Upper tracing obtained at the time of cardiac catheterization of patient J. C. Lower tracing represents pressure in the brachial artery.

SUMMARY

1. Diagnostic studies, including cardiac catheterization, are presented in four cases of congenital heart disease: patent ductus arteriosus; interatrial septal defect; interventricular septal defect; and the tetralogy of Fallot.

2. The magnitude of the shunt is calculated in each case, as is the increased pulmonary blood flow in those patients with a left-to-right shunt.

CONGENITAL MALFORMATIONS OF THE HEART AND GREAT VESSELS BENEFITED BY SURGERY*

A BRIEF REVIEW

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NEW ORLEANS

In 1902 Sherman[1] remarked, "The road to the heart is only two or three centimeters in a direct line, but it has taken surgery nearly 2400 years to travel it." There is some truth in this statement, but substitute "science" for surgery and the whole truth will be told. The surgeon was slow in travelling the short distance because physics, chemistry, anatomy, physiology, roentgenology, and pathology were slow in preparing the way for him. The great work done during the past decade must be credited to pediatrics as much as to surgery. In dealing with congenital malformations of the heart, the surgeon bears the full responsibility for the patient only during the time required to do the operative procedure.

An intelligent appraisal of a patient with a congenital cardiovascular defect can be made best by a team of specialists from the departments of roentgenology, physiology, physiochemistry, pediatrics, cardiology, and surgery. The pooling of knowledge means a more complete study of the patient, a more accurate diagnosis, and continued progress in the surgical therapy. The close association between pediatrics and surgery made it possible for Gross and Hubbard[2] to accomplish the first successful therapy.

The establishment and running of a clinic and laboratory, with adequate facilities, is an expensive project. Not many institutions can afford to spend the sum required for special equipment and also support the experimental work that is necessary for progress. In a community having several institutions with partially equipped departments, better efficiency can be had by pooling their resources in one central clinic.

Only those defects that can be relieved by present day surgery will receive brief attention. A short description of these anomalies, their principal diagnostic features, and some idea of our experience, or lack of experience, with them in this community will be given.

COARCTATION OF THE AORTA

Coarctation of the aorta may be defined as partial or complete constriction occurring somewhere in the arch from the origin of the subclavian artery to or even beyond the ductus arteriosus. Bonnett[3] recognized two distinct types: the infantile type and the adult type. In the infantile type there is a broad constriction proximal to the ductus arteriosus. The ductus remains patent and, depending upon the degree of occlusion of the arch, carries a major portion or all of the blood that reaches the lower extremities from the pulmonary artery to the descending aorta. In this anomaly, death usually occurs during the first year of life. In the adult type, regardless of the degree of constriction of the lumen of the aorta, the ductus arteriosus undergoes normal obliteration. Thus, the blood that reaches the descending aorta arrives there through the collateral channels of the chest wall, the shoulder girdle, and the internal mammary artery. Death usually occurs before the age of forty.[4] Both types occur more frequently in the male.

We shall concern ourselves with clinical identification of the adult type, because it can be relieved by surgery. Taussig[5] describes the appearance of the patient as striking. The individuals are well developed, sturdy in appearance, and have an early onset of puberty. Cyanosis and clubbing of the fingers are not present.

The pulse pressure is decreased in areas that receive blood principally or entirely through collateral channels. Pulsations of the vessels of the lower extremities might

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be feeble or absent. The left radial pulse will be diminished when the coarctation involves the left subclavian artery. Abnormal pulsations, palpable and visible, are noted in the cervical and thoracic areas where the collaterals have undergone dilatation. Dilatation of the ascending aorta simulates aneurysm of that organ, but absence of the tracheal tug will assist in differentiating the lesions.

Hypertension is a common finding. The brachial blood pressure is considerably higher than the femoral pressure. The difference is mainly in the systolic pressures, causing the pulse pressure of the lower extremities to be much lower than that of the upper extremities.

Examination of the heart usually fails to reveal any important change from normal.

X-ray examination of the chest shows notching of the ribs, a most characteristic finding. Diodrast[6] may be used to visualize the obstruction in the arch; this is not usually necessary.

Crafoord and Nylin[7] (1944) were the first to excise the constricted segment and do an end-to-end anastomosis in the human. Gross[8] followed shortly after (1945) with the same type of technic. He has called our attention to two important technical points; first, immediate replacement of excessive blood loss; second, the gradual release of the obstructing clamps on the aorta, to avoid a too rapid rush of blood to the lower part of the body. Failure to observe the latter might result in immediate death.

Blalock and Park[9] have suggested that the left subclavian artery be used to shunt blood past the constricted portion when it is impossible to excise and rejoin the ends. Whenever it can be done, excision is the method of choice.

Childhood is the optimum period for surgery of this anomaly. In this period the tissues are more elastic, sclerosis has not yet made its usual early apperance, and the collaterals are not as difficult to deal with.

We have seen but one patient with this anomaly. The patient was a 35 year old male with severe hypertension and advanced arteriosclerosis. Both Blalock and Gross advised against surgery in this instance.

VASCULAR RING

Certain abnormalities in the devolpment of the aortic arch and its branches result in patterns that cause sufficient pressure upon the trachea and esophagus to produce grave symptoms and endanger life. The predominating symptoms caused by the vascular ringlike constrictors are dysphagia and stridor, which appear shortly after birth and persist. Swallowing usually initiates stridor and cyanosis, especially in the recumbent position. A history of repeated upper respiratory infection and pneumonia is common.

Once a vascular ring is suspected, x-ray examination with barium will reveal the defect. Notches in the esophagus at the site of the constricting vessels will show in anterior-posterior and lateral views.

Surgical therapy consists of dividing the vessel that causes pressure or the one that causes counterpressure, whichever is the smaller or more accessible. Treatment should be instituted early to avoid death from pneumonia.[10]

Our experience is limited to one eighteen month old patient who had a double aortic arch. Both limbs of the arch were of equal size. There was also a patent ductus arteriosus connecting the left limb with the pulmonary artery. This patient did not survive the surgery done.

PATENT DUCTUS ARTERIOSUS

Patency of the ductus arteriosus is normal during fetal life, being essential for the by-passing of blood from the pulmonary artery to the aorta. After birth an open ductus is no longer necessary, so obliteration occurs as a normal process. Complete obliteration might not take place for several months or a year. Patency can be considered permanent if it is present at the end of the second year. Persistent patency after birth might be essential to life, as in individuals who have the extreme tetralogy of Fallot. Since the ductus arteriosus is the only channel for the blood to reach the

lungs, life is not possible after its obliteration.

Abbott[11] found that this malformation occurs two to three times more often in females than in males.

The diagnosis of patent ductus arteriosus is not usually difficult. There may be some difficulty encountered in early infancy and after cardiac failure occurs. Gross[10] and Ware have pointed out that "a patent ductus arteriosus can be recognized (or excluded) by a few minutes of intelligent use of the stethoscope."

One of the most characteristic features of uncomplicated patency is the lack of subjective symptoms. Only a very wide patency of the ductus produces symptoms in the absence of heart failure and other complications. A large number of diagnoses are made during routine examinations. Lack of subjective symptoms often plays an important role in delaying surgical closure of the shunt. The parents, and too often the pediatrician, are reluctant to submit the aparently healthy child to surgery, but this delay invites complications that change a relatively simple operation into a difficult one and decrease the patient's chance for complete recovery.

In contrast to the symptoms, the physical signs are striking. There may be stunting of growth, depending upon the degree of patency. The continuous machiery murmur is the most characteristic finding. It is heard all over the anterior chest, but is maximum in the second interspace to the left of the sternum. In this latter location a thrill of varying intensity is also palpable. The murmur is transmitted to the neck and to the interscapular area.

The size of the heart depends upon the volume of blood that passes through the shunt. The greater the quantity the greater is the load placed upon the heart and the earlier cardiac enlargement will begin. A wide shunt also causes a decrease in diastolic pressure with consequent widening of the pulse pressure. Under such circumstances the pulse assumes the Corrigan type.

The findings at x-ray and fluoroscopic examination vary, but commonly there will be cardiac enlargement, prominence of the pulmonary conus, the hilar dance, and increased vascular markings in the lung fields.

The treatment is surgical closure by single or multiple ligatures as used by Gross and Hubbard[2] in 1938 in the first successful closure ever done, or by division and closure of the divided ends, as Gross now recommends.[10]

The indications for operation may be stated simply, the presence of a patent ductus arteriosus in a child over 2 years. This is especially true if there is cardiac enlargement or evidence of endocarditis and/or endarteritis.

In August 1939,[12] the first closure was done at Charity Hospital in New Orleans. There have been only 18 patients operated upon at Charity Hospital, Touro Infirmary, and Lakeshore Hospital during almost ten years. These were done by several surgeons. There were 3 deaths—all from massive hemorrhage. One patient has been observed for nearly ten years and another for eight years. Both patients have lived normal active lives. The first has had two deliveries without showing any cardiac symptoms. The second was operated upon after spending many months in bed because of "heart disease". She has had no illnesses since ligation was done and she is living a very active life.

The prognosis after surgery is good. Patients who do not have complications develop normally and enjoy unrestricted activity. Those who have cardiac enlargement will show regression of cardiac size, some hearts returning to normal size. Marked improvement is also noted in patients with bacterial endocarditis and endarteritis.

TETRALOGY OF FALLOT

The tetralogy of Fallot is a congenital malformation of the heart that presents four anatomical variations from the normal; namely, pulmonary stenosis or atresia, dextroposition of the aorta, intraventricular septal defect, and hypertrophy of the

right ventricle. Taussig[5] points out that the combination with pulmonary stenosis is a different clinical entity from that with pulmonary atresia. She uses the term "tetralogy of Fallot" for the anomaly with stenosis and "extreme tetralogy of Fallot" for the other with pulmonary atresia. She places the distinction principally on a funtional basis.

In the tetralogy of Fallot the blood follows a normal path from the right ventricle to the lungs and life is possible without a patent ductus arteriosus. Patients with this anomaly may live many years, depending upon the degree of pulmonary stenosis.

In the extreme tetralogy of Fallot there is atresia of the pulmonary artery, so blood can reach the lungs only through the aorta and ductus arteriosus. Life is sustained only so long as the ductus remains open. There is usually a delay in obliteration, six months to one year, but death follows rapid progression of symptoms and signs in about one year. It is imperative that surgery be done very early if the infant is to be saved.

The tetralogy of Fallot is the most commonly encountered anomaly of the cyanotic group. The principal findings are cyanosis and clubbing of the fingers and toes. These, like most of the other clinical findings, are due to anoxemia and polycythemia (6 to 9 million).[13] Other findings are low oxygen saturation of arterial blood, ranging from 80 per cent in mild cases to 20 per cent in severe cases; dyspnea on exertion; stunting of growth, weak pulse; and low pulse pressure.

The heart is small with hypertrophy of the right ventricle. There is a systolic murmur best heard to the left of the sternum. A thrill is often present at the base.

X-ray examination shows the heart to be normal in size. There is absence of fullness in the region of the pulmonary conus and narrowing of the shadow cast by the great vessels. The arch of the aorta might be on the right side. The lung fields are clear, but shadows are found at the hilus that are cast by multiple small collateral vessels.

The electrocardiogram shows marked right axis deviation and an unusually high P wave.

Tetralogy of Fallot might be confused with extreme tetralogy of Fallot, the Eisenmenger complex, pulmonary stenosis, and Taussig's syndrome.[14] The last shows intense cyanosis with relatively high oxygen saturation and fair exercise tolerance caused by complete transposition of the great vessels, a small auricular septal defect, and a greatly dilated pulmonary artery.

Patients with mild symptoms and signs may live long and useful lives. One patient, a 27 year old white female, is married and carries on her household duties without distressing symptoms. She came to the hospital only because her physician requested that she be studied. This patient needs no special therapy. However, the majority of patients with the tetralogy of Fallot are not so fortunate—treatment is usually necessary and might be urgent. The only treatment that offers relief is surgical intervention.

Taussig suggested that by adding more blood to the pulmonary arterial circulation there would be relief from anoxemia and better tolerence for exercise. Blalock,[14] following this suggestion, devised a series of shunting operations that turn systemic blood into one of the pulmonary arteries by way of the subclavians, innominates, carotids, and even the partially obliterated ductus arteriosus has been used. The carotids are the least desirable for shunting because of the cerebral complications that follow. Preservation of the subclavian, he suggests, will lessen the chance for cerebral damage.

Potts, Smith, and Gibson[15] create a direct opening between the left pulmonary artery and the aorta. By using the special clamp they have devised, one eliminates complete occlusion of the aorta, thereby preserving the circulation to the spinal cord and lower extremities during the process of making the side-to-side anastomosis. This method of creating an artificial

ductus arteriosus is particularly useful in adult patients.

Anastomosis of the great vessels is best done by the suture technic, approximating intima to intima while everting the vessel walls. Fine braided silks (0000 or 00000) are most satisfactory. Potts[16] does not believe that it is necessary to place intima to intima in joining the aorta to the pulmonary artery, because the great difference in pressure between the two vessels will eliminate the possibility of the formation of an obliterating thrombus.

Only 5 patients with the tetralogy of Fallot have been operated upon at Charity Hospital. This is a small number, when we consider the population of the area we serve. Since many children with mild to moderate degrees of stenosis present only mild cyanosis and slight polycythemia, they might be overlooked by those who are not constantly looking for congenital malformations of the heart.

SUMMARY

1. Patients with congenital malformations of the heart and great vessels are best studied by teams of specialists representing pediatrics, cardiology, roentgenology, and surgery. A special clinic with adequate laboratory facilities should be established.

2. The symptoms and signs of diagnostic significence, found in the four types of malformation that are benefited by surgery are briefly reviewed.

3. The dangers of complications have been mentioned and early operative therapy, to avoid them, stressed.

4. The importance of bearing these abnormalities in mind during routine examinations of children is also stressed.

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COMPLICATIONS OF CORONARY OCCLUSION

C. T. YANCEY, M. D.
AND
M. W. HUNTER, M. D.
MONROE

INTRODUCTION

Since the advent of anticoagulant therapy for coronary occlusion with myocardial infarction, many of the complications due to thrombo-embolism have been decreasing in incidence. We still encounter, however, certain conditions which are seen often enough to bear careful watch for and attention to. In the vast majority of instances these complications are part of the disease state while others are individual problems in the case under consideration. Needless to say, there are many rare conditions which complicate myocardial infarcts which must be dealt with from time to time, but we will discuss some of the more frequently encountered ones as to their etiology and therapy.

The following is a list of the most frequently encountered conditions found in the

Presented at the First Annual Scientific Meeting of the La. Heart Association and the Sixty-ninth Annual Meeting of the La. State Medical Society, May 7, 1949.

treatment of individuals with myocardial infarcts:

1. Thrombo-embolic
2. Arrhythmias
3. Congestive heart failure
4. Myocardial rupture
5. Diabetes mellitus

THROMBO-EMBOLISM

Since the anticoagulant therapy of coronary occulusion has been considered in so much detail at this meeting we wish only to discuss it briefly and mention some of the high lights of thrombo-embolism which have been encountered in our private cases. This represents a series of 43 cases of which 30 were treated with anticoagulants and 13 did not have this form of therapy. Three of these died suddenly and there was no opportunity to institute anticoagulant therapy.

The high incidence of thrombo-embolic complications with fatal termination or considerable morbidity has been stressed recently by Wright *et al*,[1] and at times anticoagulant therapy is not sufficient to prevent them. However, therapy with anticoagulants has lowered the incidence of fatal thrombo-embolism by about 9 per cent, from 24 per cent to 15 per cent. This probably represents a decrease in the incidence of peripheral thrombophlebitis and mural thrombi which so frequently lead to fatal embolism.

We have recently seen two instances of cerebral embolism with resultant aphasia and signs of peripheral palsy which had their origins from mural thrombi following myocardial infarcts. Both of these cases were atypical in that the signs of central nervous system involvement overshadowed the cardiac symptoms. In one instance the coronary occlusion occurred approximately one month prior to the onset of the central nervous system symptoms. In the other, the diagnosis of a cerebrovascular accident was made twenty-four hours before the electrocardiographic tracing confirmed the presence of a myocardial infarct.

In order that cerebral hemorrhage might be excluded, spinal fluid surveys were done with negative findings in each instance. Following this, anticoagulant therapy was instituted. At the time of the most recent examination both patients were convalescing satisfactorily and there were practically no signs of central nervous system residua.

Peripheral thrombophlebitis deserves some mention since prolonged bed rest and inactivity are integral parts of the accepted regimen of therapy. It may become quite a problem if anticoagulants are not used especially in the face of varicosities of the lower extremity. This, we believe, is a definite indication for anticoagulant therapy. One case of this type has been encountered where an extra two weeks of hospitalization were necessary when anticoagulants were not given initially. This patient responded satisfactorily to a combination of paravertebral blocks, penicillin, and anticoagulants.

Propagation of coronary artery thrombi is always a real hazard since an additional insult may be the one incident which turns a successfully treated case into a fatal one.

To date we have not encountered any recognizable instances of pulmonary infarcts or peripheral arterial emboli either in the patients treated with anticoagulants or by the conventional methods. Wright, Marple and Beck[1] report an incidence of 9.4 per cent in the untreated cases and 5.2 per cent in the treated group. They also note that over 50 per cent of the patients in the treated group were not receiving anticoagulants at the time of the occurrence of pulmonary infarction. Peripheral emboli were found in 3 per cent of their untreated cases and in only 1 per cent of the treated cases.

From this brief resume of thrombo-embolism encountered in coronary occlusion, it is quite evident that anticoagulants have materially lowered the incidence of this important group of complications. This is especially true when prompt diagnosis and evaluation lead to early therapy with anticoagulants.

ARRHYTMIAS

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and Jaffe[2] and Levine,[3] these will disappear spontaneously, but if they persist for long intervals, may lead to congestive heart failure and death. Accompanying conditions, such as hypertension, and cardiac enlargement, increase the gravity of the prognosis. Patients in older age groups are more likely to develop arrhythmias and according to Katz and Mintz[4] there is a higher mortality rate among females than males.

No consistent incidence of the occurrence of arrhythmias has been reported. It varies between 9 per cent and 27 per cent, but averages about 18 per cent. These irregularities may appear at any time, but the highest incidence is noted in the first two or three days of the disease.

Many observers have noted the gravity of increased heart rates on the outcome of patients with coronary occlusions. This is true both in patients who have sinus mechanism or an aberrant pacemaker. From this it is quite apparent that patients who have a normal sinus mechanism and a rate of less than 100 per minute have a much better prognosis than the ones with tachycardia.

PREMATURE BEATS

Although not usually considered a type of arrhythmia, premature beats are so often seen that they are thought of as part of the disease. Masters, *et al*,[2] reported that about 25 per cent of their patients had premature beats. These are usually ventricular in origin and represent irritable foci in or near the infarcted zones. Occasionally auricular premature beats are noted, and at times they may precede auricular fibrillation or auricular flutter. The occurence of premature beats does not alter the over-all mortality rate.

Since premature beats are so frequent, no particular therapy need be instituted, but at times, especially those cases which have symptoms and those of mulitple origin will present quite some difficulties. The usual line of approach is that of sedation in the form of barbiturates, vasodilating type of drugs, and finally either digitalis or quinidine. In our series there have been 33 cases or an incidence of 79 per cent in which premature beats were found at times during convalescence, but none of them have required any special form of therapy.

TACHYCARDIAS

Paroxysmal ventricular tachycardia is one of the most serious mechanism disturbances although it only occurs in about 3 per cent of cases according to Levine.[5] If not interrupted the person may rapidly go into shock and die of congestive heart failure. None of the usual methods of carotid sinus pressure, or ocular pressure, will stop the rapid rate. At times spontaneous cessation of the abnormality will occur. The drugs employed most often are quinidine and magnesium sulfate. Quinidine may stop the rate after administering 3 grains, but larger doses are usually necessary. Twelve to fifteen grains several times daily are at times needed to prevent the recurrences. If a person is sensitive to quinidine or this does not produce the desired effects of rate slowing then magnesium sulfate in amounts of 2.0 to 4.0 grams, intravenously, may be tried and prove successful. Digitalis is not to be used since it has no effect on slowing the rate and may even increase the rate or perpetuate it.

The one patient seen by us who developed ventricular tachycardia had spontaneous cessation of the irregularity. His convalescence was uninterrupted and to date the irregularity has not recurred.

AURICULAR FIBRILLATION AND FLUTTER

Of all the arrhythmias, auricular fibrillation probably occurs most frequently. Masters, *et al*,[2] report a 7.3 per cent incidence, while Mintz and Katz[4] observed it in 18.1 per cent of their series. The older age patients and hypertensives show a greater tendency to develop auricular fibrillation, and its onset is usually within the first week. Some patients exhibit transient episodes of fibrillation, while in others it may persist even to the point of being the basic mechanism after recovery.

Therapy is centered around digitalis with rate slowing as the chief objective to prevent the development of congestive heart failure. After the onset of congestive

failure mercurial diuretics and a salt free diet are quite beneficial. The mortality rate after the onset of auricular fibrillation is about 50 per cent with congestive failure as the chief cause of death. Such authors as Parkinson and Campbell[6] and Levine,[3] on the other hand, state that auricular fibrillation has very little influence on the prognosis.

Auricular flutter is found only in a small percentage of patients—ranging from 1 to 4 per cent of patients. The importance of this arrhythmia is the extremely high mortality rate both with and without the administration of digitalis. Congestive heart failure accounts for the death of most patients, but the side effects of digitalis cannot be exonerated in some instances where death ensues. The role of digitalis will be considered later under the discussion of congestive heart failure.

Our series of cases includes only one with auricular flutter and none had auricular fibrillation. This one patient with flutter maintained good compensation on digitalis. Attempts were made to restore normal sinus mechanism with quinidine, but this was unsuccessful. Subsequently normal mechanism was obtained. The patient died six months later quite suddenly.

CONGESTIVE HEART FAILURE

When the area of infarction is large enough, nearly all patients develop some degree of heart failure. This is usually explained on the basis of insufficient cardiac output with resultant "forward failure." As the patient improves, this will gradually rectify itself and will require no special attention. The patients will exhibit basal rales, enlarged liver, dyspnea, and edema.

Congestive failure is frequently associated with mechanism disturbances, such as auricular fibrillation, tachycardia, and heart block. Therapy directed toward rectifying these irregularities will at times control the congestive heart failure, but as has been pointed out previously, a high mortality rate is encountered. In some patients the mechanism and chronic failure persist even after apparent recovery from the acute episode and subsequently lead to the death of the patient. These patients have underlying cardiac pathology, such as coronary sclerosis, old infarcts, or cardiac enlargement. The over-all mortality rate from complicating congestive failure has generally been reported as being about 45 to 50 per cent of all cases.

Much controversy[5, 7] has existed in the past as to whether digitalis should be administered to patients who have myocardial infarcts complicated by congestive heart failure. This is predicated on the finding of increased systemic emboli with death resulting when digitalis has been given for either arrhythmias or congestive failure. Peters, *et al*,[18] pointed out that none of their patients treated with digitalis had emboli after dicumarol therapy. This seems to be a logical conclusion especially in patients who did not have auricular fibrillation prior to the acute infarction. If anticoagulant therapy is instituted early and intracardiac emboli averted then digitalis should not be contraindicated. On the other hand Masters,[2] *et al*, and Levine[5] state that digitalis may prolong or produce other more serious forms of arrhythmias. They have employed mercurial diuretics and oxygen if cyanosis or dyspnea is present, and only give digitalis if a rapid ventricular rate persists in auricular fibrillation.

Nine of our cases have had digitalis therapy with no ill effects or deaths which can be attributed to the drug. Two of these were given digitalis for congestive failure developing after the acute infarction, one for auricular flutter during the acute phase, and the remainder for congestive failure, which appeared in the first few days of the disease, from mechanism disturbances or as a result of large infarcted areas. All cases except two were over 63 years of age and had previous evidences of heart disease. Four of these nine patients have since died.

Quinidine has been used a great deal recently in the treatment of ventricular tachycardia and prophylactic use has been made of the drug when frequent premature beats are present, especially those of multiple foci. Some authors consider it super-

fluous and dangerous since ventricular tachycardia will cease spontaneously without therapy at times and one is not able to predict which cases to treat. We have not utilized quinidine routinely or in the presence of premature beats. Only one patient in this series received quinidine when he developed auricular flutter. The drug failed to restore normal mechanism, but it reverted spontaneously to normal at a later date.

HEART BLOCK

Patients with myocardial infarcts exhibiting varying degrees of heart block usually have preexisting heart disease in the form of cardiac enlargement, hypertension, previous infarctions, or congestive heart failure.

Incomplete heart block has a greater tendency to disappear and to return to normal than complete heart block. Cases of complete heart block with extremely slow ventricular rates almost invariably terminate fatally, usually as a result of the serious underlying pathology. The drugs ordinarily employed are of no avail. Intraventricular block is also associated with a serious form of cardiac derangement. Occasionally it is transient but usually persists and markedly increases the gravity of the outcome. This has been emphasized by Masters, *et al*,[2] who stress the point that septal involvement has occurred and that the block is usually permanent. This is particularly true of intraventricular block.

We have had one case of complete heart block which terminated fatally. This occurred on the sixth day of the disease while the patient was in a satisfactory stage of anticoagulant therapy. Congestive heart failure resulted and accounted for the patient's death.

MYOCARDIAL RUPTURE

Myocardial rupture has been reported often enough for it to be taken with some seriousness especially during the first week of an acute infarction. The statistical studies presented show that it occurs in 2.1[8] to 73[9] per cent of cases. Some authors report a high incidence but probably this does not represent a true cross section of cases. In general, such an incident usually occurs in the more elderly group of persons whose age averages 65 years. It practically never occurs in healed infarcts. The first week after infarction gives the highest incidence and it gradually becomes rarer during the second week. After the sixteenth day healing has progressed far enough for the condition to be a rarity.

The site of rupture is seen usually in the left ventricle and more often in the anterior than the posterior wall. Rupture takes place in the weakest portion of the infarcted area where liquefaction necrosis has taken place as a result of the infarction. This usually begins as a tear. Perforation of the intraventricular septum has been reported in isolated cases when the septum was involved by the infarction process.[10-12]

Although exertion is usually considered to be the most frequent precipitating factor, some ruptures occur when the patients are at absolute bed rest. Diaz-Rivera and Miller[13] postulate that increase in intraventricular pressure probably precipitates the tear and that this occurs in the majority of instances as a result of physical exertion. There are cases in which the entire thickness of the myocardium has been involved. Another contributing factor is that of hypertension. Edmonson and Hoxie[14] point out that persons with blood pressures over 140/90 had three times the incidence of ruptures as in normotensive persons. This, however, does not explain all instances of cardiac rupture since many patients maintain a high pressure for several days following the acute episode.

In the vast majority of cases the clinical diagnosis of cardiac rupture is not made ante mortem. It should be suspected in the patients who have a bout of sudden sharp chest pain with dyspnea and shock. Death intervenes in just a few hours and possible very soon after rupture. The rapidity of demise depends upon the size of the rupture and how soon complete cardiac tamponade occurs. Invariably this is the cause of death with this complication.

Although rupture of the intraventricular septum is rather rare, it should be diagnosed clinically in the presence of a sud-

denly developing systolic murmur which is harsh in character and accompanied by a thrill. Both of these signs are usually found in the fourth or fifth interspace just to the left of the sternum. Electrocardiagraphic tracings show evidences of acute infarction with right ventricular strain. Most cases die within a few hours but a few have withstood this insult and have lived a few months. No type of specific therapy directed toward these two conditions is of any value.

DIABETUS MELLITUS

Since coronary artery disease so frequently complicates diabetes mellitus, this combination of diseases bears consideration. It is rather infrequent that one sees diabetic coma or acidosis complicated by coronary occlusion, but if they do occur much ingenuity is necessary for treatment. It has been pointed out by Cruickshank[15] that the heart of a diabetic cannot utilize blood sugar unless insulin is added in physiologic doses. Also Raab and Rabinowitz[16] showed that immediately following coronary occlusion abnormal glucose tolerance curves could be demonstrated and that these returned to normal in 67 per cent of cases. They attributed this to disturbances in the vegetative nervous centers of the brain. Others attribute abnormal and transient glycosuria during coronary thrombois to edema of the medulla and lower pons, also to reflex spasm of already diseased pancreatic vessels.

In reviewing this problem, Granirer[17] advises that the therapy of diabetic acidosis complicated by myocardial infarction should be based upon energetic attempts at control of the acidosis, particularly in the early stages of the disease. Insulin should be given generously especially in the first six hours with normal saline by vein and additional fluids by mouth to restore fluid and electrolyte balance. Relatively high blood sugars and glycosuria can be maintained, but the chief object is to combat the acidosis, early and rapidly. Other therapy should include bed rest for an extra period of time even though further sedation is necessary. Oxygen should be given for several additional days and there is no contraindication to anticoagulant therapy.

SUMMARY AND CONCLUSIONS

1. We have attempted to review briefly the current literature relative to the more common complications of coronary occlusion and the management of each one.

2. Representative instances of each complication have been given from our series of 43 cases when such were encountered.

3. Emphasis has been placed on the advantages of anticoagulant therapy of coronary occlusion during the acute phases of the disease. The successful treatment of two cases of cerebral embolism from mural intracardiac thrombi has been discussed.

4. Premature beats were encountered thirty-three times representing 79 per cent of cases. These were asymptomatic and no specific therapy was needed. One case of auricular flutter, and one of ventricular tachycardia, and one of complete heart block were encountered. The patient with heart block died and the other two recovered.

5. Nine cases were treated with digitalis for various complications, usually congestive failure. Of these, five were under anticoagulant therapy and none of them had ill effects from the drug or recognizable embolic complications. It is our opinion that digitalis can be used safely during the acute phases of myocardial infarction when its indication arises.

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ARTHRODESIS OF THE ADULT KNEE: WITH SPECIAL REFERENCE TO CHARCOT'S JOINT*

W. HAMMOND NEWMAN, M. D.**

NEW ORLEANS

Most of the present opinion of the surgical treatment of Charcot's knee is with Stokes,[6] who states that the few optimistic reports in the literature should be viewed cautiously, that conservative treatment is the one choice, and that a few so-called successful examples of arthrodesis have become "unfused" as late as seven years. At best, this thought must be widespread for the current American literature contains few references either to success or failure of operation.

The reasons for failure are not well reviewed and only two are listed:

1. *Secondary infection.* In the past this has been the more grave, for it resulted in the absolute loss of the operation and not infrequently led to amputation as a life-saving measure.[6, 7]

2. *Inability to secure bony union between the tibia and femur.* This is the most common event, for it is present at least once in every series of cases. The cause is rather obscure, and most opinions hinge on:

a) Vascular mechanism:

Soto-Hall[3] believes the sclerosis seen in the x-ray films is due to a relative ischemia of the bone ends adjacent to the affected joint; as a result the opposing surfaces after the fusion are not sufficiently vascular to be supplied with osteogenetic properties, and a pseudarthrosis results. His answer is a two stage arthrodesis, in which the first step is a drilling operation to secure vascularity.

b) Osteogenetic mechanism:

There is, however, some doubt that the above is the principal factor because Charcot's joint is considered to be a neuropathic joint rather than a solely syphilitic joint; and is present in other conditions (especially syringomyelia). In fact, the method of its experimental production in animals is section of the sciatic nerve followed by repeated trauma. Steindler[10] gives the pathology as the exact opposite of arthritis deformans; the Charcot's joint is first proliferative, then degenerative; whereas arthritis deformans is first degenerative then proliferative. The pathology begins in the subchondral region where the sclerosis on x-ray is first manifest. This is in reality a remodeling process with osteoblastic and osteoclastic activity rearranging the internal architecture without any actual growth. Simultaneously there is cartilage growth on the joint surface and destruction in the deeper layer. This is the proliferative phase. It is followed by the degenerative, where cartilage dissolution predominates, in some places the cartilage being completely destroyed, thus hastening the osteoclastic activity that is well advanced in this stage.

Yet bone is continually formed at both phases, although in a different site: the periphery of the joint where calcification of the ligaments, capsule, and muscle takes place. The picture, then, is one of a progressive joint disintegration (the rate of which varies); however, it is a degenera-

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**From the Independent Orthopedic Service of Charity Hospital, New Orleans; service of Dr. George C. Battalora.

tion characterized by laxity rather than rigidity; and a degeneration in which bone formation is present, but minimal, and lacking in any bridging properties.

Steindler[11] and Cleveland[7] believe in operating upon these cases early, before the destructive stage is well advanced so that stability and length are maximum. Bosworth[1] grasped the difficulty with the usual technic (Hibbs), and attempted to overcome it in two ways:

1. *Maintenance of position*: This he secured by driving a three flanged nail across the junction to keep the bone ends properly aligned, and add rigidity to the structure.

2. *Firm apposition of bone ends.* After ten days the patient was ambulated, the weight bearing serving to keep the tibia and femur in contact.

This seems to be the crux of the situation. Although there is osteoblastic activity at the ends, it is at best of low caliber, and has little or no ability to bridge fibrous gaps, or to markedly strengthen bone grafts that bridge wide spaces. A diminished circulation under such cirmumstances could do nothing but augment the difficulty.

In our hands the three flanged nail did not securely imbed itself in any cortical or sclerotic bone, and as a result there was some mobility at the site of anticipated union. [Figures 2 (c); 2 (d); 3 (a)]. Furthermore, the nail is driven from the femur into the tibia, and it is technically difficult to maintain apposition during insertion. While the second part of the operation (early ambulation) may be excellent for more actively ankylosing conditions, it is begun only after enough time has elapsed to permit fibrous tissue to fill the space between the ends. Impaction only takes place during the time weight is borne on the leg, but the reverse is true the rest of the time. Fairbank[12] has shown the diurnal variation which occurs similar to this in a normal knee. Because of the minimal instability of our two knees on the table, our patients were put in spicas, which prevented the after treatment; thus the failures could not be attributed to the Bosworth method.

The purpose of this paper is to present 5 cases in which bony ankylosis was desired, yet in each case the condition is one that lends itself poorly to surgery. There are 4 cases of neuropathic syphilitic knees of which 2 cases are rearthrodesis; and 1 case of active tuberculosis of the knee. To satisfy the difficulties of the problems in adult cases the position is securely and rigidly fixed at the operation by external pin fixation and an intramedullary graft, and bony union is promoted by continued bony apposition due to the spring in the pins.[13, 17]

OPERATION

The operation now used is really a combination of two advocated by Key,[1] with the modifications that the bone graft is more in the fashion of Hatt,[2] and the osseus contaction is more like that of Charnley.[13] An exposure [Figure 1 (a)] is gained by a long-

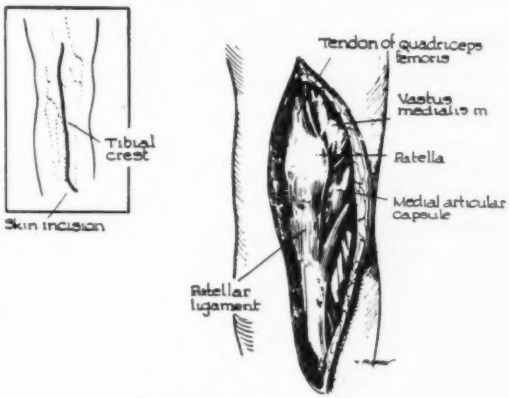

Figure 1. Procedure. (a) Incision.

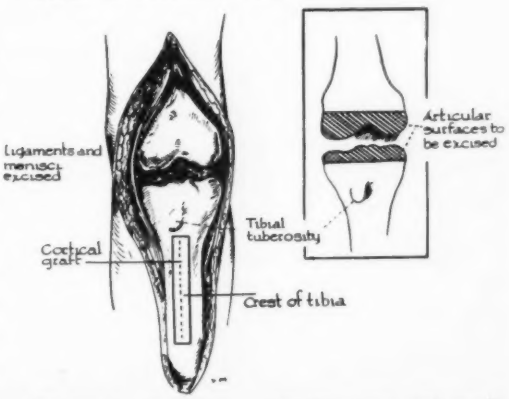

Figure 1. Procedure. (b) Exposure and graft site.

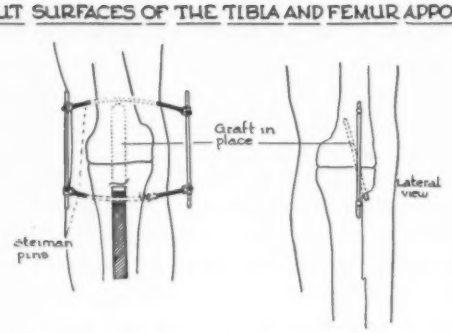

Figure 1. Procedure. (c) Final position.

itudinal incision beginning above the suprapatellar pouch, and extending over the patella, to the patella tendon, and curving laterally to avoid the tibial crest, going down to the mid-third of the tibia. The suprapatellar pouch, patella, entire synovia as far posteriorally as can be reached, fat pad, minisci, and cruciate ligaments are excised [Figure 1 (b)]. Using an amputation saw the distal femur and proximal tibia are removed just far enough from the joint to secure flat surfaces of good bone. Next the graft is taken from the crest of the tibia just below the tibial tuberosity, the length being twice the distance from the cut tibial end to below the tuberosity, or at least 3½ inches. With towel clips the wound is closed and two long 3/16 inch Steinman pins are driven transversely across the femur and tibia equidistant from the cut ends, at least one half inch longer than the length of the graft. Both ends are fitted together in the exact position and held together while an assistant springs the medial ends of the pins together and fixes them under tension with a Roger Anderson bar; after which the lateral ends are treatin a similar fashion. (Figure 1 c).

This secures a continued apposing pressure by the spring in the pins, and testing at this stage shows that the arthrodesis is stable to lateral movements and rotatory strains. There is flexion-extension present but this is stopped by driving the graft proximally in the depths of its bed across the cut surfaces. In addition the pins prevent any change in position during the driving. The wound is closed in layers and a spica is applied which is left on for six to eight weeks, followed by a long leg cast until there is radiographic evidence of union.

COMPLICATIONS

1. *Pin tract infection*: This was present in three out of the four cases in which pins were used, and started after the long leg cast was applied. It was prevented in the last case by keeping the cast especially well moulded to the thigh at all times. This difficulty may also be overcome by placing two pins in the femur, so that stability of the cast may be maintained. [Figures 3 (c) and 4 (d)].

2. *Fracture of the graft*: During the change of the spica in the first case the graft was fractured in spite of attempts to prevent this. It is fortunate that the site was within the tibia, and that apposition was not lost, and fusion resulted. Since the use of the pins this has not been present again. [Figures 2 (e) and (f)].

3. *Fracture of the tibia*: It must be kept in mind that apparent spontaneous fracture may be the first sign of a neuropathic extremity, and in a series quoted by Steindler[9] of 89 cases fractures happened in 28 femurs, 16 tibias, and 14 femoral necks. He further points out that this is more like the fractures of Paget's, that is, a transverse fracture through chalky, porotic bone. On the other hand Bishop, Stauffer and Swenson[14] had fractures 10 times in the tibia at donor sites; and King[15] had it happen 5 times in 100 grafts. Because our grafts are massive, and the site in our case is the upper end of the donor area, it is believed this is a fracture of the latter type and must be considered a disadvantage in this method. [Figure 4 (d) and (e)].

4. *Posterior tibial nerve palsy.* While a tourniquet was used in each case, a transient, incomplete palsy occurred only in 1 case, and this was one in which the graft perforated the posterior femoral cortex. [Figure 6 (c)].

ARTHRODESIS TIME

1. Case No. 1—7 1/2 months (no compression used)

2. Case No. 2—5 months (Cases 1 and 2 are re-fusions)

3. Case No. 3—5 1/2 months

4. Case No. 4—6 months (questionable union)

5. Case No. 5—5 months (T. E. not Charcot's)

The average time in these cases is 5 3/4 months.

CASE REPORTS

Case No. 1. A. J., aged 55, colored male, right knee. Pain, swelling and instability were present and increasing for "several years" (time indefinite), with only vague references to trauma. On November 4, 1947, Bosworth type of fusion was attempted, but because of instability a refusion was done May 28, 1948, in which instance no compression was used, and the graft was fractured two weeks postoperatively during spica change. Healing was uneventful and weight bearing was begun January 5, 1949, with no subsequent complications.

Figure 2. Case A. J. (b) Original lateral view

Figure 2. Case A. J. (a) Original anterio-posterior view

Figure 2. Case A. J. (c) Following nail arthrodesis.

Figure 2. Case A. J. (d) Following nail arthrodesis. The nail has retracted and tibiofemoral apposition is not maintained.

Case No. 2. I. S., aged 44, colored male, right knee. For five years prior to admission this patient had noticed swelling of the right knee. His main complaint was that the knee was becoming progressively unstable. On December 2, 1947, a Bosworth type of arthrodesis was attempted, which was followed by progressive extrusion of the nail and separation of the surfaces. October 12, 1948, rearthrodesis. March 23, 1949, weight bearing with no further complications.

Case No. 3. A. W., aged 60, colored female, right knee. One year prior to admission this patient had injured her knee. At the time there was not much difficulty; however, about six weeks postinjury she began to have progressive pain, swelling, and difficulty on weight bearing. October 1, 1948, arthrodesis; March 10, 1949, weight bearing; April 26, 1949, fracture of the tibia through the donor site, which healed in four months.

Case No. 4. W. B., aged 59, colored male, right knee. In 1940 during a hospitalization for gastric

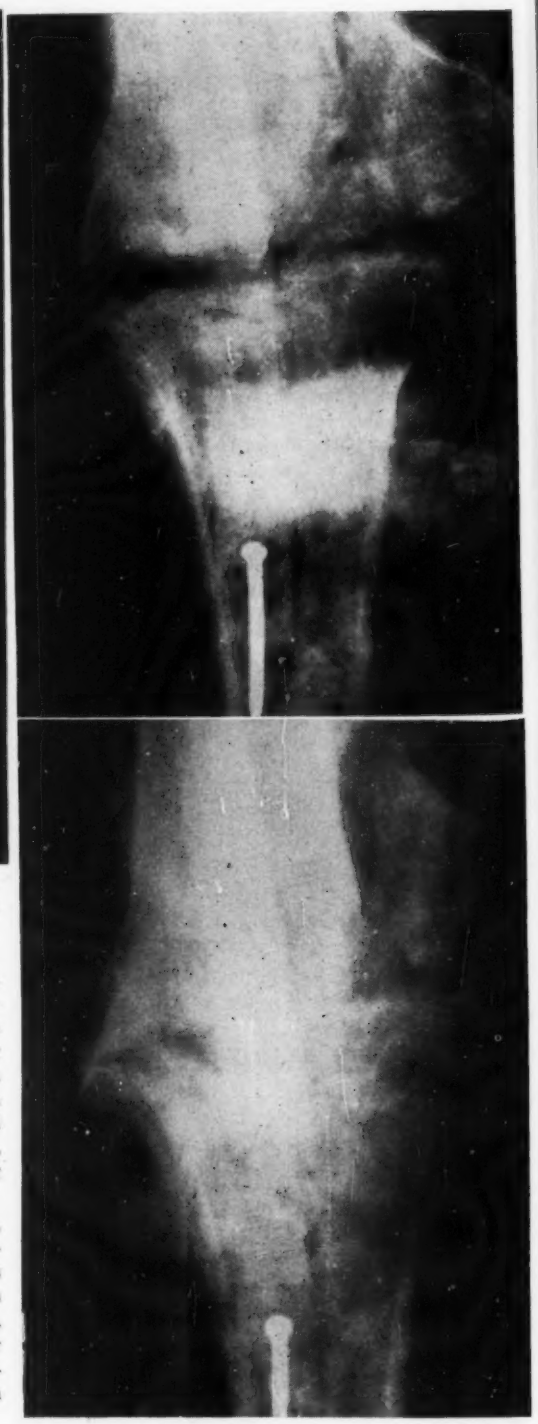

Figure 2. Case A. J. (e) and (f) Refusion. Fracture of graft within substance of tubia.

Figure 2. Case A. J. (g) and (h) Anteroposterior and lateral views seven and a half months later. Solid union.

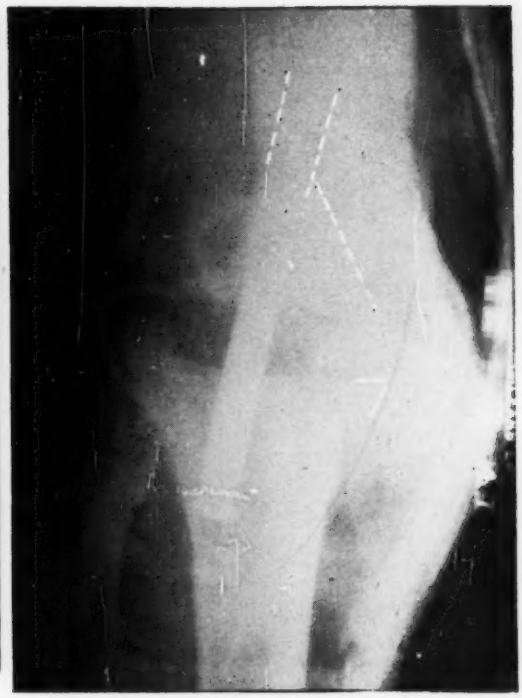

Figure 3. Case I. S. (a) Anteroposterior view after nail arthrodesis; no bony contact, and nail retracted.

Figure 3. Case I. S. (b) Anteroposterior five months after rearthrodesis; solid union.

Figure 3. Case I. S. (c) Lateral. Note pin tract in femur.

Figure 4. CaseA. W. (b) Anteroposterior five and a half months after arthrodesis.

Figure 4. Case A. W. (a) Preoperative anteroposterior view.

Figure 4. Case A. W. (c) Same date, lateral view. Note pintract in femur.

Figure 4. Case A. W. (d) Anteroposterior view four months after above. Note union of tibial fracture.

Figure 5. Case W. B. (a) Preoperative anteroposterior.

Figure 4. Case A. W. (e) Lateral view. Site of fracture is upper end of graft donor area, yet knee is still solid.

Figure 5. Case W. B. (b) Preoperative lateral view.

Figure 5. Case W. B. (c) Postoperative anteroposterior four months later. There is only questionable union.

Figure 5. Case W. B. (d) Postoperative lateral view four months later. There is only questionable union.

symptoms a diagnosis of syphilis was made for which he received sporadic treatment. In 1943 he injured his knee logging and in 1945 he had a medial meniscectomy with only symptomatic progression of pain and swelling. January 4, 1949, a fusion was done. July 6, 1949, there was questionable solidity. The pins were removed but a long leg cast was left on. Shortly thereafter the patient died in another hospital, of a condition not related to the operation.

It is interesting to note that specimen of each of the cases above showed bone debris in the synovial membrane as reported by Horowitz,[16] although only in the last was the lesion early enough to be considered of diagnostic importance.

Case No. 5. J. B., aged 29, colored male, right knee. For six years beginning at the age of 12 this patient had polyarthritic joint swellings lasting three or four months, involving various joints in combination with the right knee. At the age of 18 this subsided. At about 27 an insidious pain and swelling affected the right knee, of which a definite diagnosis could not be made, At 28, a synovial biopsy was performed, but the wound did not heal. The cultures were inconsistent for about

Figure 6. Case J. B. (a) Preoperative anteroposterior view.

Figure 6. Case J. B. (b) Preoperative lateral view.

Figure 6. Case J. B. (c) Postoperative anteroposterior view five months later. Small cystic area in medial femoral condyle cultured T. B. at operation.

Figure 6. Case J. B. (d) Postoperative lateral view. Note graft perforates posterior femoral cortex. Patient had incomplete posterior tibial nerve palsy for three weeks postoperatively.

six months, then suddenly showed tubercle bacilli (even on direct smear). In conjunction with streptomycin, an arthrodesis was done April 12, 1949, with union September 14, 1949. Full unsupported weight bearing was started six weeks later.

SUMMARY AND CONCLUSIONS

1. Charcot's knee is a joint which can be treated by arthrodesis or rearthrodesis.
2. A method is presented which will accomplish this, utilizing the principles of the rigid fixation to all strains and of active contaction to promote osteogenesis.
3. Arthrodesis time averaged 5 3/4 months.

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CONGENITAL MEGACOLON (HIRSCHSPRUNG'S DISEASE)

REPORT OF TWO CASES IN EARLY INFANCY

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NEW ORLEANS

AND

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The first definitive description of congenital megacolon was presented by Hirschsprung in 1889;[1] however, Bright[2] is credited with its earlier recognition. Congenital megacolon has been described as becoming manifest in the neonatal period; its recognition has become clearer in this age group in recent years.[3]

It is not the purpose of this paper to review the literature of congenital megacolon, but to present 2 cases wherein the condition was felt to be present in the early weeks of life.

REPORT OF CASES

Case No. 1. E. F., a 9 week old, white male infant was admitted with a complaint of fever and "swollen abdomen". The mother noted that his abdomen had been prominent since birth, and bowel movements could be induced only with cathartics. One week prior to admission, he began vomiting his formula, became severely obstipated and his temperature varied between 101° and 103° F. (R). Since that time, the fever persisted, and he vomited intermittently after many feedings. Small, firm pieces of stool could be obtained only by repeated enemas. There was no hematemesis or melena.

Past history revealed that the patient was delivered by cesarean section, and weighed 8 lbs. 12 ounces. He had been perfectly well except for the presenting complaints.

The maternal history is of interest, in that, there were six spontaneous abortions, and one full term stillborn preceding the birth of this infant. There were no living siblings.

Physical examination at the time of admission revealed a well developed, fairly well nourished white male infant, acutely ill, with a tremendously distended abdomen of doughy consistency. The temperature wos 102° F. (R) and the pulse was 110 beats per minute. He weighed 9 lbs. 15 ounces. The heart and lungs were negative. There were no abdominal masses palpable, and borborygmi were absent. Digital rectal examination was negative, and a small amount of watery stool was expelled on the gloved finger.

The blood picture disclosed a hemoglobin of 9.2 gm., erythrocytes 4.2 million, and leucocytes 5,250 with 47 per cent polymorphonuclear cells and 53 per cent lymphocytes. Microscopic examination of a centrifuged urine specimen showed 3 to 4 granular casts with 4 to 5 leucocytes per high power field; subsequent urinalyses revealed approximately the same findings. The serologic tests for syphilis, examination of the stools for ova, parasites, and cysts, as well as stool cultures were negative.

Erect and supine roentgenograms of the abdomen exhibited several gas capped fluid levels, apparently in the dilated large intestine (Fig. 1). A tentative diagnosis of intestinal obstruction, due to congenital bands or volvulus was made, and an exploratory laparotomy was performed twelve hours after admission.

At surgery a markedly distended, thick walled large intestine extending from the ileocecal valve to the anus was found, without evidence of an extrinsic or intrinsic obstructing lesion. Owing to the extreme degree of distention, deflation of the intestine was accomplished by use of a 25 gauge needle. The abdominal wound was repaired. At this time it was felt that the severe degree of thickening of the intestinal wall, together with the dilatation, was in keeping with a diagnosis of megacolon.

The postoperative course was uneventful, except for the first four days following surgery, during which time the temperature spiked daily to 104° F. (R). During the first week, the abdomen

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Fig. 1—Case E. F. Erect roentgenogram of abdomen showing dilated intestine and gas capped fluid levels.

was soft and normal; soft yellow bowel movements were passed spontaneously. Parenteral fluids, supportive whole blood transfusions, and penicillin were administered in conventional amounts.

On the eighth postoperative day the patient failed to have any stools, despite the use of mineral oil on each of several successive days. Prostigmine hydrochloride (1:4000, minims 4, every four hours) was begun on the thirteenth day and the response was gratifying with one to three soft yellow stools passed daily. This dosage was slowly reduced to minims 4, every eight hours during the next week.

Because of the favorable results obtained with prostigmine, it was our hope that active peristalsis would persist after its discontinuance. After four days of spontaneous, normal appearing bowel movements, it became necessary to resort to prostigmine hydrobromide, 15 mgm., daily, orally, because of recurrence of constipation.

At 13 weeks of age (four weeks after admission), he began to vomit all of his feedings, and a profuse diarrhea developed. Prostigmine was discontinued, and parenteral fluid therapy was instituted with prompt cessation of the diarrhea. Two days later normal bowel movements occurred.

Prostigmine hydrobromide (15 mgm. daily) was re-instituted, with the desired results, and he was discharged on this medication at 14 weeks of age, with symptomatic "cure".

Second Admission:

One week following discharge, he was readmitted with vomiting and abdominal distention. There had been no bowel movements for three days prior to this time.

Physical examination revealed an acutely ill, moderately dehydrated, dyspneic infant, with marked abdominal distention. Peristaltic sounds were audible and outline of the large intestine was clearly visible. Auscultation of the lungs disclosed increased breath sounds with numerous coarse inspiratory and expiratory rales bilaterally.

Roentgenograms following barium enema revealed no evidence of narrowing or obstructive lesion of the large intestine (Fig. 2).

Fig. 2—Case E. F. Oblique view of barium enema, prior to surgery.

The carbon dioxide combining power was 18 vol. per cent and the serum chlorides, 544 mgm. per cent. Complete hemograms and urinalyses were of no diagnostic significance.

The dehydration and acidosis were corrected by intravenous fluids, and the pneumonitis, complicated by secondary infection, was treated with

penicillin, sulfadiazine, and streptomycin. The vomiting persisted and his rapid downhill course precluded further diagnostic procedures.

Prostigmine hydrobromide and hydrochloride failed to elicit bowel movements. Acetyl-beta methycholine hydrobromide (mecholyl), in doses up to 300 mgm., daily, by mouth, was unavailing. The distention persisted and did not respond to enemas, rectal tubes, Wangensteen suction, and finally, spinal anesthesia (25 mgm. novocaine).

Erect roentgenograms of the abdomen at this time revealed marked distention of the upper intestinal tract and stomach. (Fig. 3). It was our

Fig. 3—Case E. F. Supine view showing marked distention of stomach and upper gastrointestinal tract.

feeling that a small intestinal obstruction was present in conjunction with the congenital megacolon; however, surgery was impossible because of his critical condition. He expired on the fifth day of his second admission.

Post-mortem Examination:

The important pathologic observations were limited to the thoracic and abdominal cavities. There was patchy atelectasis present in all lobes of the lungs. Microscopically, frothy, pink material was found in some of the alveoli together with many macrophages.

The peritoneal cavity contained approximately 200 cc. of serofibrinous exudate. The jejunum in the first 30 centimeters was dilated one and one-half times normal size, with numerous small petechiae in the mucosa. Where the dilatation ended, an adhesive band constricted the jejunum to a point of partial obstruction, the lumen of which would admit a lead pencil. Organized adhesions were observed between the obstruction and the transverse colon. The large bowel appeared markedly thickened and dilated throughout, with an edematous mucosa. The lymphoid tissue was hyperplastic. Microscopically, the small bowel showed edema of the mucosa and submucosa with marked congestion of the submucosa; an associated chronic inflammatory reaction was present. The serosa of the large bowel was thickened and the muscular portion edematous, with thickening of the longitudinal and circular layers. A slight round cell infiltration was present in the submucosa with discrete foci of lymphocytes. Occasional small foreign body granulomata were noted in the subperitoneal connective tissue; small crystals of talc were identified in these granulations. The myenteric plexus was identified and it appeared histologicaly normal.

It was the pathologist's opinion that the obstruction of the jejunum was the major cause of death coupled with a megacolon of the congenital type.

Case No. 2. A premature negro male, weighing 1990 grams, one of twins, was the result of the 11th pregnancy which terminated in a breech delivery after a six hour labor. Respiratory distress became noticeable shortly after birth. During the first ten days of life the respiratory difficulties gradually subsided with adequate oxygen and careful handling.

Owing to the absence of meconium stools a digital examination was performed on the second day yielding a small amount of semiliquid meconium and flatus. No spontaneous stools occurred until three days later; these appeared normal in color and consistency. Abdominal distention had been a prominent feature since birth.

Eight days postnatally, bloody mucus, apparently due to a rectal fissure, appeared in the stool and persisted for two weeks. Following this, the infant became quite listless and lethargic and passed an occasional small amount of loose stool. The response to parenteral fluids, whole blood transfusions and adjusted oral intakes of skimmed milk was at best, equivocal. Each attempt to reinstitute oral feedings, was followed by extreme abdominal distention necessitating further parenteral fluid.

Blood electrolytes were maintained within normal range with adequate intravenous fluids. Stool cultures on two occasions were negative. *Pseudomonas aerogenes* and *Proteus vulgaris* were recovered in moderate growth from one stool culture. The serologic test for syphilis of both mother and

infant was negative by the complement-fixation and flocculation methods. Blood cultures taken on two successive occasions remained sterile. Anemia was at no time a serious consideration, although frequent supportive small blood transfusions were administered. Penicillin was begun at 35 days of age in conventional dosage and continued for 10 days without noticeable effect.

At the age of 49 days roentgenograms of the abdomen revealed tremendous dilatation of the large bowel. (Fig. 4). Following this procedure

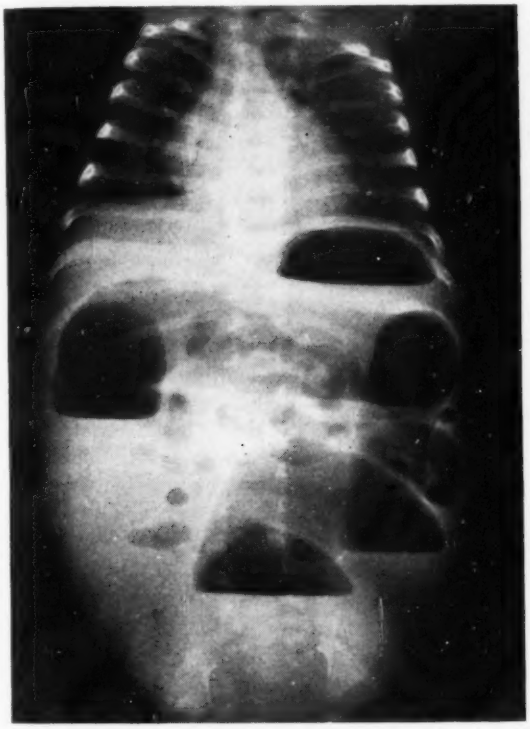

Fig. 4—Case 2. Supine roentgenogram showing extreme degree of dilation of large bowel in the premature infant.

the infant entered an irreversible shocklike state and expired.

Postmortem Examination:

The pertinent findings were limited to the thoracic and abdominal cavities. The lungs bilaterally showed scattered areas of the nonexpansion type atelectasis. Microscopically, the interstitial tissue was infiltrated with lymphocytes and macroghages.

In the abdominal cavity, scattered fibrous adhesions between the gallbladder and transverse colon were prominent. The large intestine appeared tremendously dilated from the ileocecal valve to the anus. No evidence of rectosigmoidal narrowing or anorectal stricture could be determined. The dilated colon measured 2 centimeters in its transverse diameter, and the wall was pale and edematous. A mucosal ulceration, 0.1 centimeter in diameter, was found in the first portion of the ascending colon, approximately 4 centimeters above the cecum. The microscopic examination revealed edema of the serosa with infiltration by lymphocytes and plasma cells. The ganglion cells between the muscle layers showed a ground glass appearance with vacuolization suggestive of a degenerative process.

COMMENT

The characteristic features of gaseous abdominal distention, intractable constipation, and visible peristalsis, as described by de Takats and Biggs,[4] Muesham[5] and Lockhart-Mummery[6] were present in these cases. This latter author noted, in his series of 100 cases, symptoms frequently had their onset within the first days or weeks of life. It is possible that symptoms of megacolon may be produced by congenital anorectal stricture; these can be relieved by repeated rectal dilatations and are not to be confused with congenital megacolon of Hirschsprung, (Brennemann)[7]

According to Swenson et al,[3] the use of the oblique roentgenographic projection may show an area of narrowing of the rectosigmoid. Such narrowings are frequently overlooked in routine anterior posterior projections with contrast media. In our case with an oblique view of the barium enema no point of narrowing was visible. (Fig. 2, Case E. F.)

The presence of volvulus of the elongated sigmoid loop may call for immediate surgery. (de Takats and Biggs).[4] In the first case, surgical intervention was thought necessary on this basis; however, no obstruction or stricture was found.

SUMMARY

Two cases of congenital megacolon (Hirschsprung's disease) have demonstrated the early onset of this condition, as well as the ease with which it may be confused with acute intestinal obstruction.

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DYSFUNCTION OF THE TEMPOROMANDIBULAR JOINT*

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Otolaryngologists, dentists and general practitioners often encounter patients who complain of such symptoms as head pain, vertigo, various degrees of hearing impairment, glossodynia, burning sensations of the tongue, or abnormal dryness of the mouth. As might be expected, multiple etiologic factors are likely to be involved in such a multiplicity of symptoms. On the other hand, a possible explanation, which in our experience has been not too infrequent, is dysfunction of the temporomandibular joints.

This is not an explanation which is very often considered. Indeed, unless the anatomy and physiology of these joints are clearly understood, it is likely to be received with considerable skepticism. If, however, certain basic facts are borne in mind, the association of the symptoms listed with temporomandibular dysfunction will begin to seem entirely reasonable:

1. The temporomandibular joints are in close proximity to the auriculotemporal nerves and their capsular branches, the *chordae tympanae*, the bony portion of the eustachian tubes, and the tympanic plate of the temporal bones. The development of so-called neighborhood symptoms can, therefore, be expected when these joints do not function properly.

2. Movement of these joints, which are the only movable joints in the head, is accomplished by the action of the muscles which open and close the mouth. For the purposes of this discussion the masseters and the internal pterygoids are the important muscles.

3. Normal joint function is dependent upon a normal relationship of the structures which take part in the movement of the jaws. Movement of the mandibles depends upon movement of the mandibular condyles. Movement of the mandibular condyles reflects, in turn, the occlusal relationship of the teeth, since the various movements of the mandible originate in, and return to, the state of centric occlusion. Obviously, if mandibular movements originate from an abnormal state of dental occlusion, they cannot themselves be anything but abnormal.

MALOCCLUSION AND LOSS OF VERTICAL HEIGHT

While there are various causes of malocclusion, probably the commonest is loss of one or more permanent teeth, without adequate replacement. Such losses are surprisingly frequent. The careful studies carried out by Brekhus[1] at the University of Minnesota College of Dentistry are extremely revealing to those who may not be familiar with these facts. Of the 9,450 patients, ranging in age from 10 to 75 years, who made up his material, 47 per cent had lost the lower first molar by the age of 20 years, 65 per cent by the age of 30 years, and 75 per cent by the age of 40 years. At the age of 60 years only 14 per cent of the patients still retained it.

These are significant percentages. Dentists recognize the lower first molar to be the keystone of the dental arch, because it is the first of the permanent teeth to erupt and because all the rest of the teeth are arranged around it. The loss of this tooth in early life is, therefore, often the beginning of malocclusion which manifests itself in later life in the symptoms we have listed. They are sometimes present in young persons, but characteristically they appear after the age of 40 years and they become increasingly annoying and disabling as the patient grows older.

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While the loss of the first molar is perhaps the most important dental loss, the loss of other teeth is also important. By the age of 30 years 3 per cent of the men and 8 per cent of the women in Brekhus' series had lost all of their teeth. At the age of 40 years the percentages had increased to 13 and 20 per cent. They rose progressively thereafter until at the age of 60, 56 per cent of the men and 76 per cent of the women were completely edentulous. There were also large losses of both upper and lower teeth in the patients who still had teeth in their mouths.

Some patients, of course, do not trouble to replace lost teeth, even when they have lost them all. In this group there is no question of malocclusion. There is no occlusion at all. Furthermore, the mere replacement of missing teeth with bridges or complete dentures does not necessarily end the patient's difficulties. Unless the dentures are properly made and carefully fitted, there may still be a loss of intermaxillary distance (or vertical height, as it is comonly termed), with resulting retraction of the mandibular condyles and resulting dysfunction of the temporomandibular joints. Another possibility also must not be overlooked: The human mouth changes as time passes, and often dentures which were properly constructed originally are no longer adequate because alveolar resorption has occurred.

Still another cause of loss of vertical height is the natural process of attrition. Even the person who retains all his teeth suffers some loss of substance as the years pass.

Costen[2] has well described the chain of events which comes to pass when malocclusion is present. The extent to which the jaws close is determined by the teeth. Most of the force of the contraction of the muscles of mastication is applied to the posterior third of the mandible. If there is a decrease in the normal distance between the posterior third of the mandible and the posterior third of the maxilla, the mandibular condyle on the affected side, or on both sides, will be pushed upward and backward when the mandibles are closed. The symptoms which develop, in other words, have a purely anatomic basis, which, as Costen[3] observes, is not usually considered because there are so many medical, rhinologic, and opthalmologic reasons for headache and other symptoms distributed about the ears, vertex, and occiput.

THE ORIGIN OF SYMPTOMS

Patients with loss of vertical height are likely to complain of a variety of pains in the head. Sometimes the pain is of the neuralgic type and resembles tic doloreux. Sometimes it is aching in character and is located deep in the ear. Sometimes there is no pain about the ear and the headache from which the patient suffers can be explained by spasm of the cervical muscles. This observation fits in with Wolff's[4] findings that headache may be caused by sustained contractions of the skeletal muscles of the head and neck secondary to noxious impulses arising from disease of any structure within the head. Some patients who seem to have labile vasomotor systems complain of pain of the vasodilating type, such as is seen in histamine cephalgia and in the throbbing phase of migraine. We have observed patients who showed many of the vasomotor phenomena characteristic of these conditions, including stuffiness of the nose, tearing of the affected eye with injection of the conjunctival vessels, and increased temperature and perspiration of the affected side of the face.

The glossodynia and salivary disturbances associated with dysfunction of the temporomandibular joint are explained by Costen[5] as a result of irritation of the *chorda tympani* and the auriculotemporal nerves. Affected fibers, he pointed out, which are represented by the *chorda tympani*, form the sensory portion of the facial nerve and end, in part, in the same nucleus as does the ninth (glossopharyngeal) nerve. These fibers, he continued, are considered as an aberrant strand of that nerve.

Another symptom which some patients complain of is a coppery or metallic taste in the mouth. It is not always present, and when it does occur there seems no reason-

able explanation for its presence since many of the patients who present it have no teeth at all in their mouths.

The relationship between temporomandibular joint dysfunction and loss of hearing has been widely discussed in both the medical and the dental literature. Goodfriend,[6] as a result of his studies at the University of Pennsylvania School of Medicine, concluded that malocclusion was an etiologic factor in more than 40 per cent of all deafness. Costen[2] also thinks that overbite is an important cause of deafness. Both observers[6, 7] feel, however, that if the condition is of long standing its correction, while it may produce some improvement in the hearing, is not likely to have sustained good results.

The possible causes of the hearing loss which may result from dysfunction of the temporomandibular joint include:

1. Erosion of the bony portion of the eustachian tube. Anyone who has examined Goodfriend's[6] remarkable histologic preparations taken from anatomic specimens could not doubt this cause and effect relationship. This cause was operative in at least one of our own patients, whose eustachian tubes could not be catheterized unless she opened her mouth widely.

2. Compression of the membranous portion of the eustachian tube. This cause, which Costen[5] emphasizes, arises from bulging of the internal and external pterygoids medially against the eustachian tube, combined with compression of the *tensor veli palatini* muscle. As a result of dysfunction of this muscle, the tube does not open, as it normally opens when the patient swallows. Temporary relief can be secured in this group of cases by catheterization of the eustachian tubes or by nebulization.

3. Reflex contraction of the *tensor tympani* muscle, as emphasized by Seaver.[8] Our own experience is that this is often an important factor in hearing loss and that it is analogous to the production of headache by contraction of the cervical muscles. The deafness, which is of the conductive type, is sometimes relieved by catheterization of the eustachian tube, though the relief is usually short lived.

4. Tinnitus. Our personal experience suggests that tinnitus which occurs in association with dysfunction of the temporomandibular joint has three possible causes: obstruction of the eustachian tubes; the presence of conductive deafness, which arises from spasm of the *tensor tympani* muscle; and irritation of the branches of the fifth or ninth cranial nerves. All otologists know that many patients who complain of tinnitus also complain of deafness of some degree because the noises in the head mask the sounds delivered to the ear. In many such cases, if the tinnitus can be cleared up, the deafness also improves.

While there is often a definite relationship between dysfunction of the temporomandibular joint and the development of deafness, it would be a mistake to overemphasize it. Results of treatment, it is true, are sometimes dramatic, but on the other hand, they are often disappointing in the extreme. In other words, the mere association of deafness and temporomandibular dysfunction by no means proves that the hearing loss is the result of the pathologic physiology.

DIAGNOSTIC AND THERAPEUTIC CONSIDERATIONS

We have discussed the diagnostic procedures in malocclusion at considerable length elsewhere[9] and shall not go into them here. The treatment of the condition is a dental problem, and a discussion of it is not within our province. One or two points, however, should be emphasized. The first is that when patients are encountered who complain of headache, tinnitus, vertigo, hearing difficulties, glossodynia, and salivary dysfunction, the diagnostic survey should always include an investigation of dental occlusion and of the function of the temporomandibular joint. The second is that the treatment of these cases demands the full cooperation of the otorhinolaryngologist, the general practitioner, the dentist, and the patient himself. It is essential that the patient, and all concerned with his care, should understand fully what can and what cannot be expected

in these cases. Time, effort, and money may all be expended unnecessarily if the patient seeks the advice of physicians and dentists who do not fully understand the implications of temporomandibular dysfunction. Many dentists, it seems to us, are not too understanding of either this condition or of the patient who suffers from it. They do not comprehend what Goodfriend[6] has well termed "the source of the disease factor." As an illustration, we might mention a patient of our own who was fitted with new dentures with which, when they were first used, she had a loss of 8 mm. in vertical height. Finally, we would emphasize that the dental profession has a wide field of usefulness open to it in the prevention of symptoms which arise from temporomandibular dysfunction. It is possible to prevent the development of most cases if missing teeth are replaced with proper prostheses, if dentures are properly adjusted to conform with alveolar patterns, which are constantly changing in the individual patient, and if all patients are closely watched to be certain that centric occlusion is correct.

CASE REPORTS

The following case reports, selected from our files, well illustrate the possible results of temporomandibular dysfunction. Both cases are unfortunately typical of many others, in that the basis of the patients' complaints went unrecognized for many months. Both are also typical of the excellent results which can be secured in some instances by repositioning the bite.

Case No. 1. A 56 year old white woman was admitted to the otolaryngological service of the Eye, Ear, Nose and Throat Hospital October 9, 1946, complaining of pain, loss of hearing and tinnitus in the left ear; loss of hearing in the right ear; burning sensations over the scalp; and periodic headaches in the frontal, temporal, and occipital regions. All complaints had been present for about a year. The previous history was irrelevant except for a submucous resection twenty-seven years ago and the extraction of all teeth twenty years ago. Dentures had been fitted promptly and the same sets had been worn ever since.

The resident who studied the patient could find no otolaryngological basis for her complaints. Physical examination of the ears, nose, and throat was entirely negative except for an insignificant adhesion between the middle turbinate and the septum on the left side, as the result of the submucous resection. Roentgenograms of the skull and sinuses revealed nothing at all abnormal. An opthalmologist who saw the patient in consultation could find no ocular causes for her complaints. A dental consultant recommended a new upper denture because the present plate had caused an irritative lesion of the hard palate.

When the patient obtained no relief in our clinic, she drifted away to another hospital, where she was promptly labeled psychoneurotic. This was, regrettably, the opinion of the physicians who had studied her at the Eye, Ear, Nose and Throat Hospital.

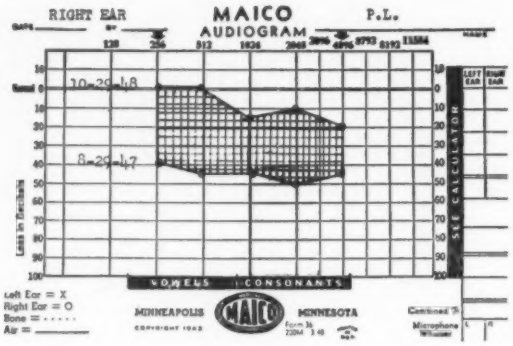
She returned to our hospital July 27, 1947. At this time she was submitted to left ethmoidectomy, which provided no relief and which was shortly followed by the appearance of true whirling vertigo. A neurological consultant ruled out an intracranial lesion and suggested Meniere's disease as an explanation of her symptoms. This diagnosis was excluded by the caloric test, which, the patient declared, produced vertigo about ten times worse than the attacks from which she was suffering. A neuropsychiatric consultation was requested but before it was secured the patient again left the clinic and made the round of other medical facilities in the city.

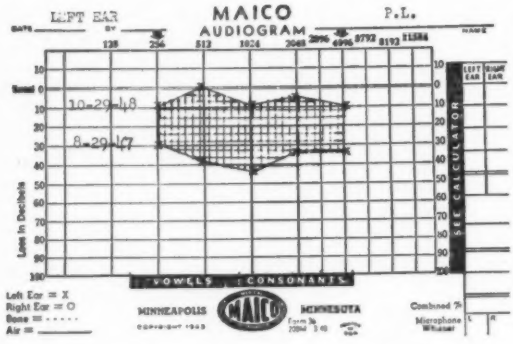
A variety of treatments gave her no relief, and she appeared again at the Eye, Ear, Nose and Throat Hospital April 26, 1948, about eighteen months after her first visit and about nine months after her second. At this time she was completely re-examined, again with negative results except that a careful dental examination revealed a loss of 9 mm. in vertical height. Her attacks of pain, which by this time had become tic-like, could be reproduced by pressure in left glenoid fossa.

A completely new set of dentures was constructed, which gave an increase of 12 mm. in vertical height. When the patient was examined July 2, 1948, eight weeks after she had begun to wear them, she reported that she was entirely free of vertigo, tinnitus, head pain, and that her hearing had considerably improved in both ears (figs. 1 and 2). She has remained asymptomatic over the ensuing sixteen months, and has obtained regular employment.

The clue to this woman's symptoms was in the possession of the physicans who first examined her but the significance of the use of dentures twenty years old was completely overlooked.

Case No. 2.—A 37 year old man was first seen July 28, 1949, five years after all of his teeth except the lower incisors had been extracted. He presented a distressing history. Although dentures had been made at once, they were never really satisfactory, in spite of numerous adjustments, and after a time he ceased to wear them. As a consequence of his almost edentulous condition he was





Figs. 1 and 2. Audiograms of the right and left ears showing auditory acuity before and after correction of loss of vertical height.

obliged to limit his diet, and as a further consequence, he lost considerable weight. He also began to suffer from a painful, burning tongue and excessive dryness of the mouth. His whole mouth was painful when he drank tart or sour liquids or ate highly seasoned foods.

The patient was an extremely sensitive, high-strung man, of Creole stock, and it was perhaps to be expected that his loss of weight, his persistently painful tongue and mouth, and the deterioration in his appearance caused by the loss of his teeth should have turned his attention inward upon himself. He gradually became unable to keep up with his work, he withdrew from society, he developed a cancer complex, and eventually he became a financial burden to his family. The various physicians whom he consulted could find nothing organically wrong with him and he received no benefit from the numerous vitamin preparations and sedatives with which he was dosed. The practitioners whom he consulted gave him such psychotherapy as lay within their abilities, but it was without effect, and after a time he was put under the care of a psychiatrist. A period of psychiatric care in a mental institution resulted in little mental improvement and in no physical improvement at all.

After the patient left the hospital he began the rounds of other physicians and finally was sent to us, with the idea of ruling out possible otolaryngologic disease. The marked dysfunction of this man's temporomandibular joints was immediately apparent and we began the routine questioning which we employ in all such cases. When we inquired whether he had ever had a metallic taste in his mouth, the question threw him, and the member of the family who was with him, into a state of great excitement. He literally shouted that he had had a coppery taste in his mouth for months, and that everybody had told him he was crazy when he said so.

Whether we were particularly lucky in asking that special question, or whether our ability to give the patient a logical explanation of his symptoms, was responsible for the liaison which we immediately established with him is hard to say. At any rate, he became entirely cooperative and was quite willing to go to the dentist and have properly fitting prostheses made. It is a matter of interest that the dentist,—who had made the original dentures with which all this man's difficulties had begun,—made the new dentures only under protest. His position was that the man was undoubtedly psychoneurotic, but since even neurotic people need teeth, he was willing to try again.

The results in this case were dramatic. As soon as the new plates were secured and adjusted, the patient began to eat solid food and gain weight. His oral symptoms promptly disappeared. As physical improvement ensued, his mental outlook changed completely. Now, at the end of three months, his rehabilitation is proceeding as satisfactorily as could be desired and we have no doubt that he can eventually return to his former work and his former status in society.

SUMMARY AND CONCLUSIONS

1. Dysfunction of the temporomandibular joint, as a result of malocclusion, may be the cause of a number of symptoms including head pain, tinnitus, vertigo, impairment of hearing, glossodynia, and abnormal dryness of the mouth.

2. Although the condition is by no means uncommon, this cause of the symptoms referable to the head and neck is generally overlooked merely because the possibility is not recollected.

3. A review of the basic anatomy of the temporomandibular joint will make clear the cause and effect relationship postulated.

4. The association occurs frequently enough to make it necessary to consider the possibility whenever a patient appears who

complains of these symptoms, although the mere association by no means establishes the diagnosis.

5. The correction of dysfunction of the temporomandibular joint resulting from malocclusion is the responsibility of the dentist, but the diagnosis is frequently the responsibility of the general practitioner or the otorhinolaryngologist.

6. The prevention of malocclusion lays open to the dentist a wide and fruitful field of preventive dental practice.

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NEW ORLEANS
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ACTIVITIES OF THE STATE SOCIETY AND ITS MEMBERS COMBATTING SOCIALIZED MEDICINE

Over an extended time, and especially during recent years, the Louisiana State Medical Society, as a corporate body and through individual members, has exerted great influence in combatting socialized medicine. The case as a whole has been presented to the public. While the issue is by no means settled, it is well to comment on the situation as it now stands and to applaud those whose untiring efforts have helped us.

The increase in dues paid by the members in recent years has greatly assisted the maintenance of these activities. People know now that there is an issue before them, and that they have much at stake. The majority of citizens now feel that it is not a battle between the "haves" and the "have nots," but a struggle for the standards of medical practice and a fight for individual freedom. The groundwork for this has been laid by the local societies. Programs in the civic interests, speeches before lay groups, talks with patients, and distribution of literature, have brought substantial citizens to our side. This understanding on the part of the public has provided a fulcrum upon which the leverage of our formal organizations could work.

The Council on Medical Service and Public Relations, under the leadership of Dr. Andrew V. Friedrichs and Dr. Max Hattaway, has been most effective in assisting local programs and providing material for a very desirable form of publicity. The service rendered by this Council has supplemented that of the local societies in presenting the matter to the public.

Another activity of great importance is that of the Committee on Public Policy and Legislation, headed now by Dr. C. G. Cole, and recently by Dr. Roy B. Harrison. This Committee has performed a most valuable service to the Society and to the public by assisting the State Board of Medical Examiners in maintaining the standards of education and practice from a legal standpoint. Changes in the practice laws which would allow practice by unqualified persons have been prevented over a period of years. The admission of cults and changes in laws which would gradually establish State Medicine have been combatted. This has been done by hard work and meticulous attention to detail.

The influence of our Society and of the public in congressional matters has been focused through the efforts of Dr. E. L. Zander, our President-elect. He and his Committee have been alert to the problems presented by bills pending in Congress. The entire delegation from the State of

Louisiana in Congress has been kept informed of the opinion of the profession. In the majority of instances they have voted with our interests in mind.

Supplementing the above, there have been many miscellaneous matters of paramount importance, which have been handled in the interest of the doctors and of the public. Our President, Dr. George Wright, and our immediate Past President, Dr. E. H. Lawson, were active in formulating the plans and pursuing the work of the Western States Medical Conference. This is a voluntary organization whose influence and aggressive measures were very largely instrumental in defeating the administration Reorganization Plan No. 1 in Congress. The effect of this plan, had it been allowed to become law, would have been to let in State Medicine by the back door. It is hoped that the same success will greet their efforts to combat the companion measure recently introduced by the administration, known as Presidential Reorganization Plan No. 27. The latter is just as undesirable as the one which was defeated in August of 1949.

Dr. O. B. Owens and his board deserve great credit for the strenuous efforts and untiring energy in pursuing the interests of the Louisiana Physicians Service. In maintaining the integrity of this organization, they are providing organized medicine with one of its most effective answers against Socialism and State Medicine. It should be a source of great gratification to the entire membership that L. P. S. is steadily increasing its field of usefulness.

The membership has cause to congratulate its committees, its spokesmen, and itself, in the success so far attained. This is, however, a cooperative effort, and our endeavors in the future must exceed those in the past.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

STATE LEGISLATIVE MATTERS

A preliminary or partial report on the progress of the Committee on Public Policy and Legislation reveals the fact that more legislation affecting organized medicine reached the legislative hopper this session than during any other session in its history. While we have been rather successful in combatting most of the adverse legislation we are not entirely over the hump and will have more battles to fight. We hope these battles will be won by organized medicine.

The most dangerous legislation we had to fight was the chiropractor bill, heard before Judiciary Committee C of the House, a committee composed entirely of laymen and a committee of the proponents' own choice. It was a hard fight but with the generous response of the members of the medical profession, including general practitioners, specialists, professors and the two deans of our respective medical schools, we weathered the storm and defeated this chiropractor bill both in the committee and on the floor of the House of Representatives.

A bill amending the osteopathic act was finally amended in committee to our entire satisfaction.

Other bills, including House Bill 896, a compulsory health insurance bill, which is a vicious piece of legislation, we hope will be defeated.

A technicians bill, with a commercial outlook and opposed by all of our pathologists and legalized technicians, we believe is or will be handled to our liking and will either be withdrawn or defeated in committee.

Many other bills are being studied and every effort on the part of your Committee is being exerted to advance them on the

calendar and secure favorable consideration for the good bills and unfavorable consideration for the bad bills.

Your Committee wishes to assure you, the members of the profession, that we appreciate your cooperation and help in contacting your respective senators and representatives, enlisting them in our behalf to defeat some of these bills so detrimental to organized medicine.

Your Committee hopes that the remaining days of this Legislature will not treat us too badly and we again thank you for your cooperation.

C. GRENES COLE, M. D., Chairman
Committee on Public Policy and
Legislation

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CONGRESSIONAL MATTERS
Reorganization Plan No. 27

The Congressional Committee of the State Society has been quite active during past weeks attempting to have Reorganization Plan No. 27, introduced by President Truman, defeated. It is the consensus of this Committee and officers of the State Society that this is a most undesirable plan and would not be for the best interest of the people of this Country. It would create a Cabinet Department of Health, Education and Security and the Plan is essentially the same as Reorganization Plan No. 1 presented last year. Last year's plan would have created a "Department of Welfare" and this year the suggested department is called a "Department of Health, Education and Security". The President has contended in his statement about the Plan that the Surgeon General and the Commissioner of Education will retain the statutory authority and duties now vested in them. He has pointed out that specific language to that effect was not used in last year's proposal, however, the fact remains that both the Surgeon General and the Commissioner of Education would be answerable to the Secretary of the proposed new department.

Opposition of the State Society is in accord with the following explanation offered by the Washington office of the AMA:

"This Plan establishes a triple instead of single department. If the Federal Government is to afford a healthful country in which its citizens may live, certainly health problems deserve the attention of a full-time agency devoted entirely to health matters. This has long been urged by our Association and recently was recommended by the Hoover Commission. The President would have us accept his suggestion that the nation's health problems be grouped in one department with educational and security activities of the government because he feels they are related. Yet it is a fair question to ask—Would it be wise to establish a combined Department of Labor, Commerce and Agriculture under a single Secretary? These obviously have a strong relationship to each other.

"This Plan provides for apointment of a Secretary of Health, Education and Security. His top assistants are an Under Secretary and an Assistant Secretary. None of these three is required to be a physician. Nor for that matter need the Surgeon General of the Public Health Service be a physician—he need only 'have professional qualifications, experience and training appropriate to the duties of his office'."

To defeat Reorganization Plan No. 27 either the House or Senate must vote to support a resolution of opposition prior to July 31 and such defeat will require 49 votes in the Senate or 218 votes in the House. Most of the members of Congress from Louisiana have stated that they are opposed to this Plan and will use their influence in attempting to have it defeated. Senator Ellender, however, has stated that "I voted for Reorganization Plan No. 1 of last year and my views in the matter have not altered since that time . . . I believe this Plan conforms with the Hoover Commission recommendations and that it is a step in the right direction toward the promotion of efficiency and economy in Government." It may be that members of the State Society can convince Senator Ellender that this Plan does not conform with recommendations of the Hoover Commission and that it should be defeated in order

that the best interest of the people of this Country may be served.

H. R. 6000

There has been continued effort in regard to opposition to H. R. 6000, the Social Security Extension Bill, the latest of which has been in regard to having the bill recommitted to the Senate Finance Committee in accordance with suggestion of Senator Harry P. Cain. Senator Cain has also presented Senate Concurrent Resolution 92 to establish a Social Security Commission to make a thorough study of the administration of the present Social Security Act. This Resolution would authorize a 16-man board to advise Congress and to provide impartial technical knowledge required by it in its deliberations; to authorize and direct an exhaustive study of the past and present operations of the Social Security Act; and to request the use of the records, services, information, facilities and personnel of departments and agencies in the government executive branch in performance of its duties. It is felt by the executive officers of the State Society that Senate Concurrent Resolution 92 should be supported and activity has been conducted in this direction.

EDWIN L. ZANDER, M. D., Chairman
Committee on Congressional Matters

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

THE AMERICAN CONGRESS OF PHYSICAL MEDICINE

The American Congress of Physical Medicine will hold its twenty-eighth annual scientific and clinical session August 28, 29, 30, 31 and September 1, 1950 inclusive, at the Hotel Statler, Boston, Massachusetts. Scientific and clinical sessions will be given on the days of August 28, 29, 30, 31 and September 1, 1950. All sessions will be open to members of the medical profession in good standing with the American Medical Association. In addition to the scientific sessions, the annual instruction seminars will be held August 28, 29, 30 ad 31. These seminars will be offered in two groups. One set of ten lectures will consist of basic subjects and attendance will be limited to physicians. One set of ten lectures will be more general in character and will be open to physicians as well as to therapists, who are registered with the American Registry of Physical Therapy Technicians or the American Occupational Therapy Association. Full information may be obtained by writing to the American Congress of Physical Medicine, 30 North Michigan Avenue, Chicago 2, Illinois.

AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians will conduct its 32nd Annual Session at St. Louis, Mo., April 9-13, inclusive, 1951. Dr. Ralph Kinsella, of St. Louis, is General Chairman responsible for local arrangements and for the program of Clinics and Panel Discussions. Dr. William S. Middleton, President of the College, Madison, Wis., is in charge of the program of Morning Lectures and afternoon General Sessions.

The 1952 Annual Session of the College will be held at Cleveland, Ohio, April 21-25, 1952.

PLASTIC SURGERY AWARD

The Foundation of the American Society of Plastic and Reconstructive Surgery offers *as its 1950 award* $500.00 (first prize of $300.00, second prize of $200.00) and a Certificate of Merit, for essays on some *original unpublished subject in plastic surgery.*

Competition shall be limited to residents in plastic surgery of recognized hospitals and to plastic surgeons who have been in such specific practice for not more than five years.

The first prize essay will appear on the program of the forthcoming annual meeting of the American Society of Plastic and Reconstructive Surgery, to be held in Mexico City, November 27-29, 1950. Essays must be in before August 15, 1950.

For full particulars write the Secretary, Dr. Clarence R. Straatsma, 66 East 79th Street, New York, N. Y.

NEWS ITEM

Doctor Roy Carl Young of Covington attended the meeting of the American Psychiatric Association and the National Association of Private Psychiatric Hospitals in Detroit from April 30 to May 5, 1950.

Doctor Young was reelected Treasurer of the National Association of Private Psychiatric Hospitals and also was elected to Fellowship in the American Psychiatric Association.

NEW FILM AGAINST STATE MEDICINE

A new film to inform the public of the accomplishments of the medical profession and point out the problems of socialized medicine, entitled *To Your Health*, has been produced for the Michigan State Medical Society by The JAM HANDY Organization. The film briefly summarizes and efectively treats a widely discussed national issue that affects the lives and well-being of every American.

Phil Faversham, son of the late matinee idol, William Faversham, heads an outstanding cast illustrating the viewpoints of people on the subject of compulsory health insurance. A reporter is sent out by his city editor to do a feature on the controversial issue of socialized medicine. He talks with the man on the street, the housewife, the worker—all who would be affected by this proposed government program.

He interviews a doctor who cites the record of the medical profession in helping reduce infant mortality, increasing life expectancy, controlling children's diseases, etc. The doctor states that more hospitals are being built, more doctors are available, more health information is being distributed, and urges people to take better care of themselves and voluntarily set up a reserve fund against ill health.

Growth of voluntary medical health funds is shown, proving that people can solve their own problems without government support. The picture goes on to point up that Federal control of medical care would make physicians servants of the state and might cause a breakdown in the present high state of efficiency among our professional men who would be under government control which is lacking in knowledge of medicine.

Finally the government is urged to help by solving some of the causes of diseases, that is, slum clearance, better working and home conditions, etc.

The film has had many effective showings in theaters in the South, East and Midwest.

WOMAN'S AUXILIARY OF THE LOUISIANA STATE MEDICAL SOCIETY

ANNUAL MEETING

The annual meeting of the Woman's Auxiliary of the Louisiana State Medical Society was held in Baton Rouge April 24-26, 1950, in conjunction with the 70th annual meeting of the Louisiana State Medical Society.

Registration began on Monday morning at the Heidelberg Hotel. The pre-convention Board meeting took place in the conference room of the Louisiana National Bank Building, Mrs. John S. Dunn presiding.

Following the meeting was a delightful luncheon at the Baton Rouge Country Club honoring Mrs. John S. Dunn. Mrs. Arthur D. Long was toastmistress.

The first session of the General Meeting was opened after the luncheon. Mrs. Wiley A. Dial, President of The Woman's Auxiliary of the East Baton Rouge Parish Medical Society, welcomed the visitors to Baton Rouge. Mrs. L. L. Davidge of Shreveport responded to this welcome in behalf of the visitors.

The reports of the Parish Presidents and of the State Committees were received. Mrs. Nathan Polmer of New Orleans reported on the Woman's Auxiliary to the American Medical Association and Mrs. Allen C. Winters of Alexandria reported on the Woman's Auxiliary to the Southern Medical Ass'n.

The second session of the general meeting was held Tuesday morning at the Woman's Club. The In Memoriam was read by Mrs. William Kohlmann of New Orleans and the Benediction was given by Rabbi Walter G. Peiser.

The Auxiliary was honored with greetings from the following guests: Dr. Daniel J. Fourrier, President of East Baton Rouge Parish Medical Society who welcomed the Auxiliary to Baton Rouge; Dr. Edwin Lawson of New Orleans, President of Louisiana State Medical Society, who complimented the Auxiliary on the cooperation and help given to the Louisiana State Medical Society. Dr. George Wright of Monroe, President Elect, Louisiana State Medical Society, who spoke of the dangerous trend in our National Government toward Socialized Medicine. Dr. M. M. Hattaway of New Orleans, Chairman of Council of Medical Services, explained the purpose and work of the Council. We regret that because of illness, Dr.

Talbot, one of our staunchest friends and Secretary Treasurer of the Louisiana State Medical Society, was unable to extend to us greetings. Mrs. David Allman of Atlantic City, N. J., President of the American Medical Association, spoke on the outstading work done by the Auxiliary, but warned that we must be ever alert. She complimented the fine work done in combating federalized medicine.

Mrs. A. A. Herold of Shreveport, President-Elect of the Auxiliary to the American Medical Association laid strong emphasis on the importance of all women registering and voting. She asked all members to do the following:

1. Know their own Auxiliary.
2. Know National Educational Campaign.
3. Know A.M.A.'s 12-point program.
4. Know your own Medical Society.

Mrs. Robert Hayes of Marshall, Missouri, President of the Auxiliary to the Southern Medical Association, reviewed the three projects which they sponsor: (1) Doctors Day; (2) Research and Romance of Medicine; (3) Jane Todd Crawford Scholarship Memorial Student Loan Fund. She invited all members to come to the convention in St. Louis, in November.

The general session continued with reports of State Committees, State Officers, and Special Committees. Then followed the election of officers. The new officers were introduced and installed by Mrs. Roy Carl Young of Covington. Mrs. DeWitt T. Milam, incoming President, asked for continued co-operation from the members.

A buffet luncheon and a style show were given at the Jefferson Country Club, honoring Mrs. David B. Allman, Mrs. Robert C. Haynes, Mrs. Arthur A. Herold. The commentator for the style show was Mrs. Richard T. Stephenson. That evening the members with their husbands attended the President's Ball at La Tropicana.

The postconvention Executive Board Meeting and school of instruction was held on Wednesday morning in the Conference room of the Louisiana National Bank Building, Mrs. DeWitt Milam presiding. Following this meeting was a luncheon at Pallud's Restaurant, honoring Mrs. DeWitt T. Milam. Mrs. F. U. Darby was toastmistress. This brought to an end a most successful convention planned by the Woman's Auxiliary to the East Baton Rouge Medical Society. Credit is due for the work done, to Mrs. John L. Beven, general chairman and her vice-chairman, Mrs. F. U. Darby and Mrs. Arthur Long.

The chairmen of the subcommittees were: Mrs. J. O. Hoth, Timekeeper; Mrs. Felix Boizelle, Registration; Mrs. B. E. Nelken, Information; Mrs. Henry W. Jolly, Jr., Publicity; Mrs. M. E. Kopfler, Transportation; Mrs. C. S. McConnell, Flowers; Mrs. T. Jeff McHugh, Luncheon No. 1; Mrs. Lionel F. Lorio, Luncheon No. 2; Mrs. J. Eugene Toups, Luncheon No. 3; Mrs. Melvin Schudmak, Printing and Tickets; Mrs. McHugh Simmons, Pages; Mrs. U. S. Hargrove, Presidents Ball; Mrs. James R. Godfrey, Style Show.

The program for this meeting was proudly dedicated to Mrs. Arthur A. Herold, President-Elect of the Woman's Auxiliary to the American Medical Association.

ORLEANS PARISH

The May meeting (and the last of the spring season) of the Orleans Parish Auxiliary was dedicated to the mothers of auxiliary members of the Parish Society, as has been the custom of many years standing. An appropriate musical program was rendered by a prominent local baritone accompanied by one of our auxiliary members at the piano.

Dr. Edwin L. Zander, recently elected President-Elect of the Louisiana State Medical Society, and a member of the national committee of the Association of American Physicians and Surgeons presented the cash awards to the winners in the essay contest of the Association which the Auxiliary sponsored in the local high schools. First prize was $50, second $25, third $10, fourth and fifth prizes $5 each, and five prizes of $1 for honorable mention.

CADDO PARISH

The Woman's Auxiliary to the Shreveport Medical Society held its May meeting on Wednesday afternoon, May 10, in the home of Mrs. T. E. Strain. Annual reports of all committees, reports of the delegates attending the state convention in Baton Rouge in April, and the installation of officers for the coming year were features of the afternoon's program.

Highlights of the many excellent reports included the report of the Bandage Center sponsored by the auxiliary in conjunction with the Cancer Society, which showed a total of 27,720 bandages made in the work room this year with 13,000 of them being used in Caddo parish. Mrs. G. A. Creel, chairman, also reported that her committee had given 926 hours to bandage work.

Mrs. C. R. Gowen reported that the auxilary had so far collected $765 for the cancer drive. The chairman of the Red Cross solicitation, Mrs. S. L. Gill reported that this drive had gone over the top and that over $6,000 had been collected for the community fund, which was 85 per cent of the quota assigned. One of the community projects of the auxiliary during the year has been the handling of all community fund raising among the medical profession, hospitals and nurses.

Mrs. C. E. Rew reported as chairman of the speakers bureau that her committee had appeared before 35 groups presenting the true picture of socialized medicine.

Mrs. L. L. Davidge, in presenting her president's report for the year, pointed out that the

Shreveport auxiliary was the first to institute a speakers bureau to provide speakers on socialized medicine to groups desiring to be informed on this subject. Mrs. A. A. Herold, Mrs. Judson Bender, and Dr. Alice Holoubek have done the speaking for the bureau. She further stated that Toy Loan Center work in which the auxiliary sponsors the Toy Loan Center at Charity Hospital and the handling of all social agency collections among the medical groups by the auxiliary were, also, two projects of the organization in addition to the regular work outlined by the national auxiliary.

The second part of the program consisted of reports of the delegates attending the state medical association convention in Baton Rouge during April. Among the delegates reporting on the meeting were the president, Mrs. L. L. Davidge, Mrs. Joe Heard, Mrs. Paul D. Abramson, and Mrs. N. Judson Bender. Mrs. Joe Heard will serve the state woman's auxiliary as first vice-president for the coming year.

Mrs. L. T. Baker next installed the officers for the coming year in a brief and impressive ceremony. Officers for 1950-51 are as follows: Mrs. N. Judson Bender, President; Mrs. W. B. Worley, President-Elect; Mrs. Paul D. Abramson, First Vice-President; Mrs. Raymond Mays, Second Vice-President; Mrs. W. M. Hall, Recording Secretary; Mrs. W. B. Brown, Corresponding Secretary; Mrs. W. J. Hill, Treasurer.

OUACHITA PARISH

Mrs. D. L. Anderson, chairman, and Mrs. Stanley Mintz, Mrs. F. E. McCarty, Mrs. C. B. Flinn and Mrs. Ralph Talbot were co-hostesses for the May luncheon of the Woman's Auxiliary to the Ouachita Parish Medical Society at a local restaurant. The U-shaped table was decorated with artistic arrangements of spring flowers from the gardens of Mrs. Armond McHenry, Mrs. R. Morgan Simonton and Mrs. Ralph Talbot. It was announced that a subscription to Ideals had been sent to Dr. J. E. McConnell from the auxiliary. The officers presented their annual report. Mrs. N. Klam announced the programs for next year. Mrs. J. E. Walsworth, Mrs. Roy Kelly, Mrs. B. J. LaCour and Mrs. Stanley Mintz were appointed by the president, Mrs. R. Morgan Simmonton, to form a budget committee.

The auxiliary voted to make a donation to the Jane Todd Crawford memorial fund. It was also voted to place racks in public waiting rooms to make available to the public literature distributed by the A.M.A. The members who had attended the recent state parties and convention in Baton Rouge reported on the meetings and parties they had attended and Mrs. DeWitt T. Milam, the state president, read a list of do's and don'ts which she had prepared for the state convention. A resolution was adopted that the auxiliary subscribe 100 per cent to the Bulletin published by the auxiliary to the A.M.A. At the conclusion of the meeting, Mrs. Simonton turned over the emblem of office to Mrs. Ralph Talbot, the incoming president.

Eight Ouachita Parish doctor's wives became officers of the Woman's Auxiliary to the Louisiana State Medical Society during the recent convention in Baton Rouge. The new officers all members of the Woman's Auxiliary to the Ouachita Parish Medical Society are:

Mrs. A. G. McHenry, State Corresponding Secreary; Mrs. DeWitt Milam, State President; Mrs. Cyril Yancey, State Treasurer; Mrs. A. D. Tisdale, Commemorations Chairman; Mrs. A. D. Scott Hamilton, Bulletin Chairman; Mrs. J. E. Walsworth, Councilor for the Fifth District; Mrs. Hollis Rogers of Winnsboro, Fifth District Chairman of Louisiana health program.

Mrs. DeWitt Milam plans to attend the annual meeting of the auxiliary to the American Medical Association, as the Louisiana presidential delegate, in San Francisco in June.

LAFAYETTE PARISH

The Lafeyette parish medical auxiliary had its annual luncheon meeting for the installation of officers with retiring president Mrs. J. B. Montgomery presiding. The new officers are: Mrs. E. W. Wynne, President; Mrs. Henry Voorhies, President-Elect; Mrs. Ralph Bourgeois, Secretary-Treasurer.

Reports of the state meeting recently held in Baton Rouge were given by two auxiliary members who attended. Mrs. Montgomery also presented a review of the auxiliary's activities during the past year.

The Lafeyette Parish Medical Auxiliary entertained the 1950 graduating senior girls at a tea at the Methodist Student Center on Saturday afternoon, April 29th.

Other guests of the Auxiliary were the wives of the dentists and druggists of Lafayette Parish. The tea was given in the interest of recruiting nurses and medical technologists.

Mrs. J. Boring Montgomery introduced the speakers, who were Miss Hermine Tate, Medical Technologist in charge of the laboratories of the Lafayette Charity Hospital; Mrs. Edward Harrell and Mrs. Stanley Morgan, both registered nurses.

The third annual fashion show of the Lafayette Parish Medical Auxiliary was staged on Thursday evening, May 18, in the Isenberg gymnasium at Cathedral High School. The purpose of these annual shows is to secure funds to help fight cancer. Members working on the committee to make this a success were: Mrs. E. W. Wynne, Mrs. J. J. Burdin, Mrs. R. H. Robinson, Mrs. Henry Voohries, Mrs. Boring Montgomery, Mrs. Richard Dalton, Mrs. Ralph Bourgeois, Mrs. Edgar Breaux, Mrs. Thomas Latiolais, Mrs. Lee Sonnier, Mrs. Zerben Bienvenu, Mrs. Tom Kimbrough.

BOOK REVIEWS

Mitchell-Nelson: *Textbook of Pediatrics*, Philadelphia, W. B. Saunders Company, 1950, fifth edition, 1658 pages, 426 illustrations, 19 in color. Price: $12.50.

This fifth edition has been completely rewritten and is now entirely up to date; many trivial errors which appeared with change of editorship in the fourth edition of this comprehensive and very useful textbook have been corrected. Particularly gratifying are the revised bibliographies strategically placed throughout the volume.

The illustrations and tables are all well done, and the general organization and indexing of the text are both excellent. Numerous improvements, with many new or rewritten sections, further serve to justify the opinion of this reviewer that this is the best quick reference source and textbook of pediatrics now available; it is amply desrving of the wide popularity it enjoys among both medical students and practicing physicians.

R. V. PLATOU, M. D.

The Diagnosis and Treatment of Adrenal Insuffiency: By George W. Thorn, with collaboration of Peter H. Forsham and Kendall Emerson, Jr. Springfield, Ill. Chas. Thomas, 1949. Illus. 171 pp.

This is a brief exposition by a man with great experience in the treatment and diagnosis of Addison's disease. Clear and explicit directions for the differential diagnosis of Addison's disease and equally clear directions for therapy are woven in with an excellent discussion of the pertinent adrenal physiology.

ALBERT SEGALOFF, M. D.

The Development of Gynecological Surgery and Instruments: By James V. Ricci, M. D. Philadelphia. The Blakiston Co., 1949. Pp. 594. Price, $12.00.

Ricci, a clinician, presents the historical background of gynecological surgery and instruments from the Hippocratic dawn to the antiseptic age. Translating and gathering facts from the original Greek, Latin, German, French, or English, the author has compiled this outstanding work from 860 references.

The ancients used iron, steel, tin, lead, copper, bronze, and occasionally gold and silver for making surgical instruments. Lucian of Samosta, about 150 A. D., compares an uneducated man owning a large library to "ignorant physicians who buy ivory medicine boxes, silver cupping glasses, and knives with gold handles, but when these instruments are needed, do not know how to use them. It is then that a really experienced practitioner comes forward with a sharp, though rusty knife, and at once relieves the patient." Thus the Hindus of the sixth century B. C. used iron and steel for instruments, but it was not until the era of Hippocrates (about 450-370 B. C.) that instruments resembling our vaginal specula and uterine dilators began to appear. Tubes for irrigating and fumigating the vagina were particularly popular.

Ricci takes us over the pus, poultice, and purifying years to the study of anatomy and development of surgery as an art. For centuries, medicine seemed to take one step forward and two backwards, but there was still progress. Gynecologists may well be proud of Ephraim McDowell who, in the backwoods of Kentucky, first removed successfully an ovarian cyst, and J. Marion Sims of Alabama, who published his article on vesicovaginal fistula in 1852.

Moritz Schuppert, surgeon to the Orthopaedic Institute, must have been in sympathy with the slaves, for in 1866 he dedicated his pamphlet on instruments which he devised for repairing vesicovaginal fistula to the "great Liberator of the nineteenth cenutry", Alexander II of Russia.

As a reference and as a book for relaxation, this excellent work is highly recommended.

EUGENE H. COUNTISS, M. D.

B.C.G. Vaccination in Theory and Practice: By K. Neville Irvine, D. M., M. A., B. Ch. Oxon. Blackwell Scientific Publications, 1949. Pp. 120. Price, 9s 6d. net.

The postwar revival of interest in B.C.G. vaccination against tuberculosis has demanded a careful review of the subject and an authoritative summary of the present status of this method of prevention. This English monograph supplies admirably the need for such a book. Beginning with a short history of vaccination against tuberculosis and of B.C.G. in particular, the chapters cover the theory of vaccination, the methods of producing the vaccine, the variations in the virulence of B.C.G. with a consideration of experiments on animals and the safety of administration of this preparation to man. The moot question as to the stability of the vaccine is thoroughly discussed. The author attempts to reconcile the earlier reports of dissociation and variations in virulence with the later absence of such reports by the theory that the virulent "S" variants have been eliminated from the cultures of B.C.G. for the past twenty years. The more difficult questions as to degree and duration of the resistance evoked by B.C.G. vaccination are gone into in detail with the conclusion that "if vaccination were to be used to replace some other antituberculosis measure, its comparative value would be of great importance; as it is employed only to supplement already existing measures, if it were to produce any degree of protection at all, its use would be justified". The practical application of B.C.G. vac-

cination and the different methods of vaccination are discussed and the worldwide use of B.C.G. up to 1948 is reviewed. The book concludes with advocacy of the use of B.C.G. in England for the protection of medical students, nurses, newborn infants of tuberculous mothers, and others necessarily exposed to infection.

The polemics of the first years after the introduction of B.C.G. have become less bitter with the passage of a generation. The foreword by Konrad Birkhaug carries over some of the crusading zeal and unwavering faith of Calmette. However, Dr. Irvine has succeeded, insofar as is humanly possible, in his aim to present an objective and impartial summarization of an important and controversial subject. JULIUS L. WILSON, M. D.

Harvey Cushing, Surgeon, Author, Artist: By Elizabeth H. Thomson. Foreword by John F. Fulton. New York, Henry Schuman, 1950. Illus. 347 Pp. Price, $4.00.

This is a short new biography, intended for the general reader and medical man. It supplements the larger work of J. F. Fulton. Harvey Cushing was born in 1869 and educated in Cleveland, went to college at Yale, and medical school at Harvard. He then became a surgical resident at the Johns Hopkins Hospital. Here he was in the right place at the right moment. Hopkins was in the vanguard of American medical schools and boasted a galaxy of unprecedented talent. Cushing worked under the great surgeon Halsted and from him not only learned meticulous technic but received the opportunity of operating at an early age; as he wrote: "People don't know what it means. Here I am a youth, doing surgical work that not one of my school confreres will hope to do for years."

Following his residency, Cushing decided to specialize in neurosurgery; here lies his chief importance to medicine. When he entered this field case material was scanty, operative mortality high, his first efforts were unsuccessful, he was discouraged by Halsted. Yet he persevered. He was instinctively an artist who drew and enjoyed art and this plus his Halsted technic were developed to a high state of perfection in a field which especially demanded it. (Watching the famous English brain surgeon Victor Horsley and others operate, young Cushing's comment had been "The technique of all these men is execrable from our standpoint.") He applied the latest discoveries in neurophysiology, which was then making strides. He insisted on autopsying and making public the results of all his cases so others would know where he stood. When he died in 1939, neurosurgery was a recognized specialty which saved lives, and a school of surgeons trained by him were operating all over the world.

Not the least of the above success was due to his attention and concern with each of his patients. Early in medical school he decided to be a "leper" and "forego social activities and devote all his attention to his work." This he did and although he had a large family . . . "he often sacrificed his family to his profession. He was almost a guest in his house . . ." All was concentrated on his patients: "Each patient became his personal responsibility, and the operation a fierce contest between him and the forces that threatened life." When he was still a medical student he thought he was the cause of the death of a patient and announced to his instructor "that he was going to leave medical school and do what he could to assist the patient's family." He was assured that he had not been responsible. The same thing may be seen towards the close of his life when he operated on General Leonard Wood who died of complications, over which no one had any control. Cushing wrote; ". . . If I had used better judgment he would certainly have been saved." His attention was rewarded by the gratitude of many patients, some of whose letters are reproduced here.

From the beginning Cushing selected what he wanted and drove hard for it. Throughout his life he was constantly "driving . . . himself, both to achieve recognition and to accomplish a little more than the next person." He early determined to become Professor of Surgery at Harvard, and turned down several offers elsewhere. The coveted chair was finally obtained in 1912. He knew the tricks of competing with others and humorously described his success to "being cagey", which meant he could draw people out, find out all they knew and then by collateral reading, know more than they did. His own life was a tremendous success story and many pages are given over to simply chronicling the honors he received. Cushing came of a line of successful American physicians and was brought up in surroundings of comparative wealth and security. Late in life he would write in an essay, "Tradition indeed is the most powerful binding influence the world knows. It lies deep in most of us, and pride in tradition supplies the glue which holds people and groups of people in cohesion. Pride in family and friends, in Alma Mater and profession, in race and birthplace, in state and nation. The controlling subconsciousness of one's stock and upbringing is something from which time and distance can never wholly wean us." As his biographer points out, this passage gives us the key to several of his own inner doors." His success was in terms of this tradition.

There are many details here of interest to medical readers. Cushing worked with the pituitary and gave his name to a disease which since has assumed importance, though it is thought to be of different nature than he originally postulated. In the field of the pituitary "his method of experiment was for the most part based on inductive reasoning. Rather than making deductions from

an assemblage of facts, he was inclined to postulate a theory and then set out to prove it." When proven wrong, he tended to cling to his ideas. However, he served to stimulate others to thinking and working on the problem. In 1902 he brought back the Riva-Rocci blood pressure apparatus, now in universal use, from Italy, and introduced it into the wards. A committee was formed at Harvard Medical School to examine it and "came to the conclusion that the skilled finger was more accurate than any pneumatic instrument and dismissed the matter as of no importance." There are many aspects of Cushing discussed which are of wide interest; Cushing the bibliophile, his travels abroad, his family and wife, his distinguished circle of friends, his contributions to the administrative side of medicine. On the whole this is a rewarding book, and in the case of the writer has stimulated interest in Dr. Fulton's larger volume. RALPH COLP, M. D.

Practical Aspects of Thyroid Disease: By George Crile, Jr., M. D., F. A. C. S. Philadelphia, W. B. Saunders Co., 1949. Illus. 355 p. Price $6.00.

In this book Dr. Crile has presented many interesting observations on thyroid disease; these observations are based chiefly upon his own experience in the surgical treatment of approximately 1000 patients, and upon several hundred hyperthyroid patients treated in the Cleveland Clinic with antithyroid drugs. Although many references are made to the extensive literature on thyroid diseases, this book is not intended to be a review or summarization of the literature.

In the first chapter Dr. Crile treats of the pertinent features of the physiology of the thyroid.

The pathology of endemic goiter is next discussed. In this chapter he uses the term "Graves disease" to describe "the type of hyperthyroidism which bears no relationship to endemic goiter, and in which the entire parenchyma of the thyroid is involved in the overproduction of thyroid hormone." In addition he uses the term "discrete adenoma" to describe a "clinical rather than a pathological entity." Throughout the book he uses the term adenoma as a nonspecific designation for nodules in the thyroid.

Subsequent chapters discuss prevention and treatment of endemic goiter; intrathoracic goiter; clinical and pathological aspects of hyperthyroidism; the diagnosis of hyperthyroidism; iodine in the treatment of hyperthyroidism; antithyroid drugs in the treatment of hyperthyroidism; roentgen therapy and radioactive iodine in the treatment of hyperthyroidism; the preoperative management of patients with hyperthyroidism; anesthesia for thyroidectomy and the anatomy of the thyroid.

As a technic for thyroidectomy Dr. Crile advocates an extracapsular ligation of the inferior thyroid arteries. The recurrent laryngeal nerves are identified if feasible, but not isolated and dissected unless a total thyroidectomy is contemplated. He then presents his excellent therapeutic results, and evaluates his own technic for thyroidectomy.

Ensuing chapters discuss complications during thyroidectomy; postoperative care; postoperative complications; recurrent hyperthyroidism; hyperthyroidism in the extremes of life; diseases associated with hyperthyroidism; and congenital abnormalities of the thyroid and thyroglossal tract.

Dr. Crile then presents a somewhat unconventional classification of the 64 malignant tumors of the thyroid he has seen from 1936 to 1945. Four types are recognized as follows: Papillary carcinoma; nonpapillary carcinoma (undifferentiated carcinoma, adenocarcinoma, angioinvasive adenoma); special types (non-encapsulated sclerosing tumor, squamous cell carcinoma); sarcoma (lymphosarcoma, fibrosarcoma).

The author presents a most timely and well reasoned discussion of statistical studies of incidence of carcinoma of the thyroid. In his series 10.9 per cent of surgically removed nontoxic nodular goiters and 24.5 per cent of surgically removed nontoxic solitary tumors of the thyroid proved to be malignant. The diagnosis of malignancy, however, was made or suspected preoperatively in 96.6 per cent of the patients with thyroid malignancy. He states that these figures could "be presented as to indicate that, (a) over 3 per cent of nontoxic solitary nodules of the thyroid are undiagnosed malignant tumors; or, (b), there is only one chance in 6675 that removal of a nontoxic solitary nodule in the thyroid will give protection against death from carcinoma of the thyroid." He feels that "the high incidence of carcinoma of the thyroid reported by surgeons is to a large measure dependent on the fact that the obviously benign nodules in the thyroid are screened out by the patient, or by the internist, and are not seen by the surgeon." He warns that solitary nodules "in the thyroid in patients of any age should be viewed with suspicion, especially (a) in children, (b) when enlarging or giving symptoms of discomfort or pressure, (c) when firm or hard, (d) or when discrete and of a different consistency from the remainder of the thyroid." He feels that "soft involutionary nodules in the thyroid, especially when multiple and not large enough to be conspicuous may be disregarded, if they are not enlarging or causing symptoms." He warns that the danger associated with a discrete nodule is not so much that it may become malignant, but that it already is malignant, and should be treated by total excision. He believes that until adequate statistical studies are available, the indications for removal of nodular goiters should be based on clinical judgment, rather than on statistical surveys.

Papillary carcinoma of the thyroid is described as a tumor of low malignancy which seldom metastasizes distantly. It is stated that "Encapsulated tumors which can be shelled out of the thyroid and which invade blood vessels should be classified as

nonpapillary carcinomas even if papillary areas are present." Concerning lateral aberrant thyroids, he states that the papillary tumors found in the lateral cervical region have in his experience, "invariably been associated with a primary tumor of the same structure in the thyroid."

Dr. Crile then discusses nonpapillary carcinoma of the thyroid, stating that there is a striking tendency for these tumors to invade venous channels, and for this reason the adjacent vessels and one internal jugular vein should be removed in the surgical extirpation of this lesion. Involvement of the cervical lymph nodes indicates lymphatic permeation of the capsule and indicates a poor prognosis. Pain and vocal cord paralysis are also very poor prognostic signs. In those patients with inoperable or metastatic nonpapillary carcinoma of the thyroid, he advises a trial of radioactive iodine if the tumor can be shown to take up iodine. He also refers to the work of Rawson in which evidence is presented that removal of all of the normal thyroid, of destroying it with radioactive iodine will in some cases stimulate the metastatic tumor to take over the function of the thyroid, and thereby be more susceptible than it was originally to the effects of radioactive iodine.

Nonencapsulated sclerosing tumors of the thyroid are then discussed. He believes these are clinically benign lesions, although he refers to them as "adeno-carcinoma, not arising in adenoma." He states that these lesions have no clinical manifestations, are usually unsuspected prior to operation, and are discovered by the pathologist. They are not encapsulated, and there is no clear cut line of demarcation betwen the lesion and surrounding thyroid. The microscopic appearance varies considerably, appearing at times like mature thyroid tissue or perhaps as solid masses of epithelial cells with little or no tendency to glandular formation. They do not infiltrate beyond the capsule or invade lymphatic or blood vessels. These lesions do not require extensive surgery or postoperative radiation.

The final chapter discusses subacute (psuedotuberculous or giant cell) thyroiditis; struma lymphomatosa (Hashimoto's thyroiditis), and Riedel's struma.

This book is well organized, and the material has on the whole, been presented with clarity. Some criticism might be made of the discussion of the pathology of thyroid disease. Apparently Dr. Crile does not believe in the pathological entity of the true thyroid adenoma (colloid, embryonal, fetal, turtle cell, etc.) and makes no attempt to differentiate these clinically from the simple involutionary nodule in the thyroid gland.

In the chapter discussing "Pre-operative Management of Hyperthyroidism," there is little or no discussion of the administration of iodine or thiourea derivatives although these topics are adequately covered in other chapters.

Dr. Crile's classification of malignancies of the thyroid, including such terms as "angioinvasive adenoma" and non-encapsulated sclerosing tumor," does little to clarify a confused subject.

Dr. Crile is to be commended particularly on his excellent discussion of the incidence of thyroid malignancy, and for especially well written chapters dealing with the surgical aspects of thyroid disease, and the evaluation of results.

This book is strongly recommended to the student of thyroid disease, and would be a worthwhile addition to the library of the general practitioner.

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